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ESSENTIALS

OF THE

PRACTICE OF MEDICINE

PREPARED ESPECIALLY FOR

STUDENTS OF MEDICINE

BY

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ARRANGED WITH QUESTIONS FOLLOWING
EACH CHAPTER

PHILADELPHIA AND LONDON
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PREFACE.

This little book aims to present no new facts, and in its composition the standard works most widely used in our medical schools have been followed.

I have tried to emphasize the more common aspects of the various diseases, to present the average case, because it seems to me that this is what the medical student should try to remember.

It is my belief that the best way for the student to learn to differentiate similar conditions is to thoughtfully work out for himself lists of the contrasting points, and I have tried to emphasize these points in the text.

To save space, it has been necessary to disregard questions of rhetoric, and while uncouthness of style may be apparent, yet clearness has, I hope, been preserved.

WILLIAM R. WILLIAMS.

Hotel San Remo,
Jan. 23, 1905.

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ESSENTIALS OF
THE PRACTICE OF MEDICINE.

INTRODUCTION.

INFLAMMATION.

To avoid frequent repetition, a sketch of the changes in inflammation is given here.

Degeneration of Cells (Epithelium, Nerve, Muscle).—When acted upon by certain poisons, the cell becomes swollen and opaque, granules (albuminous or fatty) appear, the cell may die and be detached.

Resolution.—If the cell does not die, it simply resumes its normal appearance. If it dies, it may be replaced by proliferation of similar cells, leaving the tissue as before. This change does not constitute inflammation, but it occurs in most diseases, and with inflammation.

Simple Exudative Inflammation.—1. *Congestion.*—In the affected area small blood-vessels dilate; there is increased flow of blood, but later slowing of the current; and white cells cling to the capillary and venule walls. 2. *Exudation.*—Blood elements get through the capillary walls (perhaps venules also) into surrounding tissues; red cells by diapedesis; white cells, chiefly polynuclears, by emigration; plasma by exudation (sometimes called transudation); then fibrin and serum are formed. The leukocytes usually die. In purulent inflammation there is an excess of leukocytes.

Necrotic Inflammation.—Death of tissue in a considerable mass leads to an abscess or, if on a surface, to a slough or an ulcer. Pus is an exudate composed largely of leukocytes, or this plus fragments of dead tissue.

Repair.—Resolution occurs when the tissue is restored to its original condition. The products of exudation are absorbed by lymphatics, with the help of phagocytosis. This can not occur with a mass of dead tissue, but granulation-tissue forms at the limits of necrosis, fills the cavity, changes into firm, fibrous tissue, and a scar marks the completion of healing. This scar tends to contract.

Productive or Cellular Inflammation.—Connective-tissue cells or endothelium proliferate and form masses of cells—found most typically in endothelium of blood-vessels, in pia mater, and peritoneum. It often occurs in connective tissue, and then is called interstitial inflammation (later, fibrils are deposited, cells become fewer, and firm fibrous tissue is developed and tends to contract).

Parenchymatous inflammation involves chiefly parenchymal cells of organs. They degenerate. This type occurs chiefly in glands like the kidney, liver, and pancreas. It is associated with more or less exudative inflammation.

Diffuse inflammation involves all parts of the diseased area, interstitial and parenchymatous together.

Interstitial, diffuse, and cellular inflammations do not resolve completely; the new connective tissue is a permanent addition. It tends to shrink.

In **mucous membranes** the changes are precisely analogous, but different terms are used:

(1) **Catarrhal Inflammation**: (*a*) *Acute.*—A simple exudative inflammation. There is congestion as above with lessened secretion of mucus. Exudation occurs into the stroma and on the surface, mucus is secreted excessively, epithelium degenerates and is cast off. The mixture on the surface is muco-pus, and contains mucus, white cells, perhaps red cells or pigment from them, epithelial cells, more or less serum, and often bacteria. (*b*) *Chronic.*—The above changes are protracted, and in addition there is interstitial inflammation in the stroma. There may be proliferation of the mucous glands also (hypertrophic catarrh); or the glands may become fewer, the connective tissue filling their places, and then the secretion is less (atrophic catarrh).

(2) **Necrotic Inflammation**: (*a*) *Croupous Inflammation.*—1, Congestion; 2, Exudation; 3, Necrosis of [illegible]ficial

layers of mucous membrane which forms a slough (false membrane) on the surface, composed of bacteria, epithelial cells, leukocytes, fibrin, necrotic tissue of mucous membrane. (*b*) *Diphtheritic Inflammation.*—Sometimes used as a synonym of croupous; more exactly a croupous inflammation due to diphtheria bacillus. (*c*) *Ulcerative Inflammation.*—Superficial tissues die, but are cast off at once, leaving an ulcer. Necrotic inflammation of surfaces heals by granulation. The epithelium of the surrounding parts proliferates and covers the scar-tissue. Glands are not reproduced.

An exudative inflammation may be acute or chronic; necrotic inflammation is often very acute, but may become chronic, *e. g.*, tuberculosis. Productive, cellular, and interstitial are usually chronic, but may be marked by rather acute exacerbations. Parenchymatous may be acute or chronic.

In Lymphatic Tissue.—*Hyperplasia.*—Congestion and excess of blood in the lymphatic tissue; later, swelling, often pallor (blood is squeezed out), a multiplication of lymphoid cells (lymphocytes) which may more than double the mass of the lymphoïd tissue. There may be larger round cells, supposed to be proliferated endothelium and cells of the reticulum. Resolution may now occur, or the tissue may die *en masse*; and may undergo coagulation-necrosis, or liquefy and form what is called a suppurating node. The necrosis may extend through other tissues and discharge on the surface of the body or mucous membrane. *Healing.*—Granulation and fibrous tissue form a scar and healing is accomplished. If already superficial (mucous membrane), the necrotic tissue forms a slough which is cast off, leaving an ulcer. The ulcer becomes filled by granulation-tissue, then scar-tissue, and the epithelium grows in from the edges, covering the scar, and healing is completed. Sometimes these scars tend to contract.

QUESTIONS.

Describe the changes in simple exudative inflammation of connective tissue, of mucous membrane (catarrh), of a solid gland—*e. g*, salivary gland (include resolution).

Describe necrotic inflammation in deep tissues and in mucous membranes (include repair).

Describe productive inflammation.

What is meant by parenchymatous inflammation? By diffuse inflammation?

Describe simple and necrotic inflammations of lymphoid tissue.

DISINFECTION AND ISOLATION.

Isolation within a House.—The sick-room should be on the top floor or in one end of the flat; sunshine is helpful; all furniture and hangings that can be spared should be removed; a liberal supply of fresh air is important, but it should not pass through other rooms after leaving the sick-room. Sheets are often hung over doorways, and kept moist with water to catch dust, or with carbolic or other disinfectant solution, which is probably no more effective than water. The room should be dusted with moist cloths, and these disinfected. In general only the nurse and physician should ever go into the room.

Attendants and *physician* wear washable gowns and footwear. Daily sponging of the whole body with Labarraque's solution (5 per cent. in water) is recommended by some. Before mingling with other persons they should change their outer garments. The hands when soiled should be scrubbed with soapsuds, and perhaps with 0.1 per cent. bichlorid.

Soiled clothing, bed-linen, and *dust-cloths* should be put into a receptacle kept in the room containing carbolic acid (5 per cent. in water). Green soap (2 per cent.) is added with advantage. After soaking in this for an hour or longer they may be removed, but should be kept separate in the laundry, and boiled for half an hour.

Dishes should be reserved for exclusive use in the sick-room, and put in carbolic (5 per cent.) for an hour. Any food remnants should be burned.

Excreta.—Urine and feces are received into a receptacle containing a disinfectant. Later enough more of the disinfectant to equal the volume of the excreta is added. These are thoroughly mixed and put aside for an hour, and then may be put into the sewer. Disinfectants: (*a*) fresh chloride of lime (1 per cent. in water), or (*b*) milk of lime (freshly slaked lime 12.5 per cent. in water). These mixtures do not keep, and must be made daily. (*c*) Formaldehyd (0.5 per cent.); (*d*) mercury bichlorid (0.1 per cent.), not so good; (*e*) carbolic acid (5 per cent.).

Sputum and *nasal* discharges are received on gauze handkerchiefs, which should never be allowed to dry, and should be burned. *Sputum cups:* Paper ones are burned; otherwise have carbolic acid (5 per cent.) in the cup (this must have at least an hour to act).

Patient's Body.—Desquamating skin is sponged with glycerin (10 per cent. in water). Some use carbolic acid (2 per cent.) or bichlorid (0.1 per cent.), or anoint the body with petrolatum. This is done daily. Buttocks and perineum after every stool are washed carefully with soap and mopped with bichlorid solution (0.1 per cent.) and *dried.* It is probable that any disinfectant solution which is allowed to dry irritates the skin more than it does the microbes. The custom is very general, however.

After the Disease.—The patient, if alive, is bathed and shampooed and clad in clothing that has not been contaminated. If dead, the body is sponged with bichlorid (0.1 per cent.); all orifices of body are plugged with non-absorbent cotton. The funeral should be private and very prompt.

For the *nurse*, a bath and fresh clothing throughout.

The Room.—All things are left in the room and so disposed as to allow of free contact with air; blankets are unfolded and spread on racks or chairs; closet doors and all drawers are widely opened. Stuff all cracks about windows and doors with plain cotton or paste over them several layers of paper, and fumigate.

Fumigation. (1) *Sulphur.*—Put a tub in the middle of the room, pour in four inches of *hot* water; support a tin pan on two bricks; put in the pan 3 pounds of sulphur for every 1000 cubic feet of air space to be fumigated; pour alcohol on the sulphur, light it, and leave the room at once; seal the cracks of the door of exit. After twenty-four hours enter the room, open windows and air for a day or two.

(2) *Formaldehyd.*—Use 40 per cent. solution in water and special generator. For every 1000 cubic feet of space use one pound of solution. Paraform (1 gram per cubic foot) in special candles may be used instead of this.. After six hours the room is opened and aired. Probably both these gases

act better with water. The tub of hot water gives this in one case, and the generator provides for it in the other. They kill most of the bacteria on the surfaces, not spores, but the gases do not penetrate even through many layers of cloth. Subsequent treatment is necessary.

Walls.—If papered, repaper; or rub down with bread and burn the crumbs. If not papered, wash with bichlorid (0.1 per cent.); painting is very good. Wood-work should be washed as above and repainted or varnished or polished.

Furniture.—Carpets, rugs, and blankets are best treated at a sterilizing station by steam under pressure. Bright sunshine for several days is an excellent germicide.

Mattresses.—Burn the filling and boil the cover for one-half hour or send to disinfecting station for steam treatment.

The degree of isolation and the things to be disinfected vary with different diseases. These will be learned later. Extreme care is necessary, and the physician must give the matter his personal attention.

QUESTIONS.

Give details for isolating a contagious case in a house.

Give details for disinfecting a room and its contents after a very contagious disease.

How would you disinfect contaminated excreta, bed-clothes, dishes, food, the patient's body?

FEVER.

Fever is a group of symptoms that is common to very many diseases, and renders the patient liable to certain complicating conditions that very often are due to a secondary infection with the omnipresent septic organisms. In any given disease certain symptoms are apt to be prominent and to give that disease a more or less striking character. These will be the symptoms to remember in trying to make diagnoses. If, then, the student learns thoroughly the symptoms of fever in general, he may focus his mind on the special symptoms in studying any disease, and so learn the disease with greater ease and with much greater accuracy.

A fever is apt to start in one of two ways: (1) A gradual disturbance of health, impaired appetite, constipation, probably a slight rise of temperature in the evening, headache, languor, yawning and stretching, aches in the back and limbs, a progressing feeling of weakness, and occasionally chilliness. These continue to increase until the patient feels ill enough to stay in bed, and he presents the picture of a well-marked fever. (2) It may begin abruptly; perhaps after a few hours of languor and general discomfort, the patient has a sudden chill in which the hands, feet, and lips are cold and blue; he feels very cold and shivers: his teeth chatter; perhaps there are nausea and vomiting; pulse rapid and small; rectal temperature elevated (101°–104° F.), skin temperature low (97° F. or less); the urine is increased and dilute; there is prostration. These last for a few minutes or an hour or two, and then rapidly change to the symptoms of the hot stage.

During the course of the fever the chill or chilliness may be repeated. In a young child, instead of the chill there may be a convulsion.

In a distinct fever the skin is flushed, hot and dry; tongue coated with whitish fur; there are thirst, a dry mouth, nausea, vomiting, constipation, overacting rapid heart, full bounding pulse, accelerated breathing, elevated temperature which fluctuates, highest in the evening and 1 to 3 degrees F. lower in the morning; frontal, vertical, or general headache, often throbbing; apathy; an active delirium comes on, at first nocturnal only, later it may be continuous; sleeplessness, restlessness. Urine lessened in amount, dark, heavy, very acid, with excess of urates, often a little albumin, some hyaline or granular casts. Aching of muscles, bones, and joints. Progressive prostration and some loss of weight. This condition is called a *sthenic fever.* If the fever lasts long enough or is severe enough, symptoms of more marked exhaustion and poisoning of the entire body develop. Such a state is an *asthenic fever,* or the *typhoid state.* Its symptoms are: Skin dry or clammy, pale; fingers and toes cold and perhaps blue; lips, gums, and teeth covered with a pasty or dry stuff called sordes; tongue tremulous, dry, brown, cracked, swollen; swallowing is difficult and may be possible for liquids only; perhaps a regurgitant vomiting; constipation or incontinence of feces; more or less tympanites; heart more rapid and weaker; pulse small and very soft; breathing rapid and shallow, usually through mouth; temperature high; unconsciousness; a low muttering delirium; little, restless, or disturbed sleep, or none at all; sensation dulled; blindness and deafness; tremor of fingers and toes; subsultus tendinum; constant picking at bedclothes in a purposeless way; emaciation and prostration are progressive and marked. Bed-sores may appear. Urine is retained or passed involuntarily. *Coma vigil* is a modification of this, in which the patient lies supine with widely opened eyes, but blind and in practical coma.

The fever may subside gradually, the temperature zigzags down until it reaches normal in two or three days or a week or more, all the other symptoms also disappear, periods of calm alternate with the delirium, the weight may begin to increase, and by the time the temperature is normal the patient may feel quite normal but very weak. This is defervescence by *lysis.* The fever may go rapidly by *crisis:* there are sweating, sound sleep, diuresis, and the patient awakens feeling quite normal but very weak. Within twelve to twenty-four hours the temperature falls from 103° F. or higher to normal or subnormal, the heart and breathing reach normal rates, the mind clears. Occasionally during the crisis the patient goes into collapse and may even die. Afterward the patient may remember little or nothing of the period of marked fever. The hair may fall out, but grows again; ridges on the nails appear.

If he dies, the heart may gradually weaken and stop, usually there is coma also; or there may be a steady rise of temperature for a day or two to 106°–109° F., the patient dying with *hyperpyrexia.*

Complications.—Parotitis—secondary infection from the mouth, with tendency to suppurate; it comes on late. Acute gastritis, enteritis, colitis, and diarrhea may be early or late.

Heart: Sudden death; the heart suddenly gets fast, irregular, and weak, and stops within a few minutes or hours. This is apt to occur late in the fever or in convalescence. Acute endocarditis. Thrombosis of veins, less commonly of arteries, comes on when the heart has grown weak. The blood shows an advancing anemia. Catarrhal or, less often, croupous inflammation of mucous membranes of upper air-passages is quite common at any stage. Bronchitis; hypostatic congestion of lungs (when heart weakens and the patient lies continuously in one position); pneumonia, either lobar or broncho-. Abscess or gangrene of lung. Pleurisies of any type. These occur at any stage, but are more common late.

Temperature: Remissions vary in extent and regularity. There may be inverse temperature, higher in morning, lower at evening. An abrupt fall of temperature may be associated with marked sweating. Chills or chilliness are often associated with a distinct rise of temperature. Nervous symptoms may be most marked without any meningitis. A real meningitis may rarely be found. Neuritis, single or multiple, is rather common as a late symptom, even in late convalescence. *Special senses:* Almost any inflammation of the eye may occur. Otitis media is very common. *Kidneys:* The febrile urine with albumin and casts is due to degeneration of renal epithelium. This is not usually recognized as a complication. There may be an actual nephritis, which appears during the height of the disease or in convalescence. Noma rarely occurs.

Treatment in General.—The patient should be kept in a sunny, well-ventilated room, freed from all unnecessary persons, furniture, and decorations. The bed narrow, rather high; a comfortable spring and mattress; a rubber sheet under the cotton sheet. Only enough coverings to keep the patient from feeling cold. Temperature, pulse, and respiration are recorded every three or four hours. Usually a bed-pan should be used for stools and urine. Skin sponged daily for cleanliness. To avoid bed-sores, the bony prominences, especially of the sacral region, hips, elbows, and heels, should be rubbed two or three times a day with talcum and alcohol. If the skin gets red and tender, an annular air-cushion or pillows should be placed so as to remove the pressure, and the position of the patient should be changed regularly every couple of hours. If bed-sores develop, the patient may need to be put on a fluid bed (air or water) and the sores dressed surgically. The mouth should be very carefully cleaned with a swab and some of the alkaline antiseptic mixtures after feeding. Dry, cracked lips need an ointment; for the dry tongue 10 per cent. of glycerin is often added to the mouth-wash. *Diet:* Liquids, 4–8 ounces, every two hours. Milk alone or with water, carbonated water, lime-water, or sodium bicarbonate; or it may be peptonized or variously fermented, Broths, meat juice, white of egg and water, or strained gruels may be used. Water should be given regularly. Ordinarily a patient takes two to three quarts of liquid a day. Bowels should move every day or two; enemas are very often needed. Usually cathartics also are permissible. After a stool the perineum should be carefully washed clean, and *dried.* The heart may need stimulation—whisky or brandy, 2 ounces or more a day; strychnin, etc. To avoid hypostatic congestion, stimulate the heart carefully, and have the patient lie in different positions. Temperature is best controlled by baths. Antipyretic drugs are seldom indicated. Headache and restlessness may be relieved by cold to the head, by phenacetin, etc., or opium; sleeplessness by baths or hypnotics. Nothing should be allowed to interrupt a *sound* sleep. A stuporous condition needs stimulation and food, which should be given at regular intervals night and day.

During a chill the patient should be warmly covered; a hot-water bottle put at his feet; and perhaps some alcohol may be given.

QUESTIONS.

Give symptoms at onset, during the course, and at defervescence of a mild, a moderate, and a protracted fever.

Give symptoms of a chill, a sthenic fever, the typhoid state, a crisis, a lysis.

Describe febrile urine.

What are the complications encountered in fevers?

Give the general treatment of a febrile patient.

INFECTIOUS DISEASES.

INFECTIOUS diseases are those caused, or assumed to be caused, by living micro-organisms.

The classification is not definite. In some most typical, clinically, the germ is not known; *e. g.*, measles. Others may be less typical but are sometimes included; *e. g.*, muscular rheumatism.

Stages.—*Incubation* is the time from lodging of the cause to the appearance of active symptoms. Often the last day or two of this period are marked by slight symptoms of ill health; these are called *prodromal symptoms.* The *febrile* or *active stage* is variously subdivided in some diseases, such as cholera, yellow fever, and the exanthemata.

Exanthem is properly a rash occurring in the course of a disease. It is used also to denote a disease that has a characteristic rash. The active stage is divided into (1) *invasion* (from appearance of active symptoms to appearance of the characteristic rash); and (2) *eruptive period* (during the active rash). In many (3) *desquamation,* which may extend into convalescence. This classification is not so important as formerly, and the lists of different authorities vary. All include measles, rötheln, scarlatina, varicella, variola, typhus, and most add typhoid. Sometimes erysipelas, dengue, zoster, etc., are included.

QUESTIONS.

What is an infectious disease?

What is meant by incubation period; by period of invasion in exanthemata, prodomal stage, period of eruption?

What are the principal exanthematous diseases?

TYPHOID FEVER (Enteric Fever).

Etiology.—Bacillus typhosus (Eberth). It is of moderate size, flagellated and actively motile; grows on usual media (including potato); grows best at 25°–35° C.; lives from a few days to several months in water, soil contaminated with sewage, on oysters, or dry. It is not killed by freezing; it is killed by 60° C. in twenty minutes, boiling in two minutes, by close contact with antiseptics properly used for stools (p. 20) within one-half hour. Within the body it occurs in intestinal contents and walls, mesenteric lymph-nodes, spleen, urine, blood, and therefore occasionally in any organ; in the rose spots. *Infection* is by mouth from stools or urine of typhoid patient, water contaminated directly by excreta, or from contaminated soil. This water is drunk, mixed with food, used to wash utensils (milk cans, etc.); or may contaminate oysters kept in it. Fingers or flies or dust may carry bacteria from stools or urine to food or dishes. Possibly bacteria may be inhaled and cause the disease. It occurs in epidemics; is endemic in most towns; and there may be sporadic cases of undetermined source.

Predisposing Factors.—Usually it is a primary disease. Age: usually fifteen to thirty-five, but no age is exempt. Nurses and laundresses are very liable if not careful. Armies in camp are great victims; also towns that mix sewage with drinking-water. More cases occur in late summer and autumn.

Immunity.—Some individuals are naturally immune. A second attack is quite rare. Injection of sterilized typhoid cultures seems to give some artificial immunity.

Lesions.—The lymphoid tissue of the intestine, solitary follicles, and Peyer's patches of the lower part of the ileum, often of the upper part of the colon also, is inflamed. (1) *Stage of infiltration:* There is marked congestion for a few days; then the follicles become much swollen and elevated and pale. Microscopically, there is hyperplasia of lymph-tissue, with typhoid bacilli in the lesion. This stage corresponds to the first eight to ten days of fever. Resolution, in mildest cases only, now occurs. The cells degenerate and are absorbed, and the tissue becomes as it was before the typhoid. Most cases go on to (2) *sloughing.* The follicle undergoes coagulation-necrosis, and a grayish slough is formed, which is gradually separated and cast off. This stage lasts to the end of the third week of fever. (3) *Ulcerative stage:* the ulcer left by the slough is opposite the mesentery, and is round (solitary follicle or part of Peyer's patch) or elliptical (Peyer's patch) with long axis lengthwise of gut; the edges may be undermined at first, the floor is in the submucous, or muscular, or even serous coat. (4) *Healing* begins when the slough starts to separate. There is cicatrization of the area of the ulcer; epithelium grows over it from the periphery and healing is complete. This marks the close of the fever. The scars do not constrict the gut. Mesenteric lymph-nodes are congested, then hyperplastic and swollen, and contain typhoid bacilli; resolution takes place during defervescence. The spleen is enlarged, soft, almost fluid when cut, and very dark; microscopically it shows hyperplasia and typhoid bacilli; resolution occurs during defervescence. There is associated a catarrh of the mucous membrane. The above lesions are characteristic and almost invariably present.

Less common are the following, due also to typhoid bacilli: Intestinal hemorrhage in first week from intense congestion or during stage of sloughing from an eroded vein or artery. Perforation of gut in stage of sloughing. Mesenteric nodes may necrose and rupture. The spleen may rupture. *Peritonitis:* General, from perforation, or rupture of mesenteric node; or local, from extension of inflammation through the wall of the gut, or from a mesenteric node or spleen. The lesions of the blood and the complications are mentioned with the symptoms.

Symptoms.—First a typical, uncomplicated four weeks' typhoid will be considered.

Incubation usually lasts two weeks (one to four weeks). Prodroma are usual, and last three or four days. They are headache, anorexia, languor. They gradually increase and the patient goes to bed. The onset is reckoned from this date. There are the symptoms of a fever steadily growing worse. The striking symptoms are "typhoid tongue" (coated in middle, red at tip and edges), anorexia, discomfort, tenderness and gurgling in the right iliac region. At the end of the week the spleen may be felt. There are symptoms of a slight bronchitis and nose-bleed. The temperature shows a staircase rise to the maximum (103°-105° F.) at the end of the week. Headache persists to the end of the week; there may be some delirium. There are the urine of fever, and increasing prostration. The stage lasts a week. *Second week:* A marked fever changing toward the end of the week from a sthenic fever to the typhoid state. Diarrhea appears with four or five pea-soup stools a day, in which typhoid bacilli are found. Gurgling and tenderness in the right iliac region and tympanites increase. The heart-rate is 90 to 110 per minute; the headache disappears. The blood shows Widal's reaction.[1] Dulness and delirium are more marked; the temperature continues high with little remission. Urine: diazo reaction appears,[2] and typhoid bacilli appear in it. Loss of weight begins to be marked. The *eruption* is a roseola. There is a pink papule 2–4 mm. in diameter that disappears temporarily on pressure. It develops in a few hours, lasts about two to four days, and leaves a discolored spot for two or three days more. The rash comes first on the front of the abdomen, or the sides of the trunk or on the back; spreads over the trunk and may reach the limbs. It appears from the seventh to the tenth day, lasts one, two, or three weeks; has several crops overlapping one another in time. It varies from a few spots to a profuse rash, regardless of the severity of the disease. Desquamation is usually absent. *Third week:* The typical typhoid state (p. 23) is present; diarrhea is more marked, emaciation is marked, bed-sores are apt to appear; there are the rash, palpable spleen, and diazo reaction. The temperature shows more marked remissions, which may reach 3 or 4 degrees F. at the end of the week. The daily maximum remains the same, or is a degree less. *Fourth week:* Defervescence occurs by a slow lysis, the morning remissions are more and more marked, and the evening temperature is less every day; the appetite returns; all the symptoms become quite normal along with the temperature at the end of the week.

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[1] *Widal's Reaction.*—Blood-serum is diluted with 35–50 volumes of water or salt solution. A loop of this is mixed with a loop of a young bouillon culture or other emulsion of typhoid bacilli, in a hanging drop over a hollowed slide. The bacilli collect in clumps and become motionless in fifteen to sixty minutes. This appears first on the fifth to the ninth day, and persists for a few weeks or months. It is found in the vast majority of cases.

[2] *Diazo Reaction of Urine.*—Solution (I.) contains HCl 5 parts; water to 100 parts; and sulphanilic acid to saturation. Solution (II.) contains sodium nitrite 0.5 per cent. in water. They are kept in separate bottles. Take 40 parts of I. and 1 part of II. and shake. *Mix* equal parts of this mixture and of urine in a test-tube, and float ammonia-water on top. At line of contact a cherry-red ring appears if the test is positive. This reaction usually appears in typhoid from the fifth to the fourteenth day, and disappears at end of the third week. It is found sometimes in other infectious fevers.

Special Symptoms and Complications.—*Skin:* Sudamina, at any time, associated with sweating. Abscesses and boils during fever and in convalescence. Bed-sores may appear in the typhoid state, but are usually preventable.

Parotiditis, a secondary septic infection from the mouth, is rare. There are swelling, pain, tenderness, perhaps redness over the parotid region, a temperature 1 or 2 degrees higher, and leukocytosis. It may resolve or go on to suppuration and necrosis, burrow extensively, or rupture externally, or kill. *Stomach:* At onset, intense epigastric pain, repeated nausea, and vomiting may occur, but are apt to subside in a few days. *Tympanites* may be extreme, especially in a severe case. It causes abdominal pain and discomfort, a distended, tense abdomen, obliteration of the dulness of the liver and spleen; rapid, weak heart action; and rapid, shallow thoracic breathing.

Intestinal hemorrhage occurs in 5 per cent. of cases. There may be a small hemorrhage the first week (from congestion). Usually it appears from the middle of the second week to defervescence (from erosion of vessel in slough), varies from a few drops to a pint or two, and may be single or repeated. Symptoms: One small hemorrhage causes no symptoms except blood in the stools. Many small hemorrhages cause also progressive anemia, weakness; and perhaps death in syncope. One large hemorrhage causes sudden pallor, a rapid weak heart, very small pulse, a sudden drop of temperature (2 to 5 degrees), and more or less collapse; perhaps syncope and death. After a few minutes to several hours bloody stools are passed, varying from bright-red liquid to a tarry mass. The next day the temperature resumes the usual curve.

Perforation (about 3 per cent. of cases) occurs usually in the third or fourth week, sometimes before, often later, and, as a rule, in cases otherwise severe. Symptoms: Sudden, sharp, severe pain, often paroxysmal, in the right lower quadrant of the abdomen; there are often marked sweating; rapid, feeble heart action; shallow, rapid thoracic breathing; a sudden fall of temperature (2 to 5 degrees); great prostration, often followed by a rally of strength; spasm and rigidity of abdominal muscles; obliteration of hepatic and splenic dulness at once or in a few hours; sometimes dulness in the flanks (liquid from the intestine); and tenderness, which may be general, or local and deep. *Peritonitis* develops after a few hours with clammy skin, sweating, a pinched expression, vomiting, constipation, usually great abdominal distention (sometimes this is strikingly absent), no liver or splenic dulness. There may be increasing dulness in the flanks, peritoneal crepitus, or gurgling; leukocytosis rapidly develops; there are a rapid, weak heart, a running pulse, shallow thoracic breathing; the fever steadily rises. Finally, there are mental depression, coma, advancing prostration, high temperature, and death in one to three days. The symptoms may be masked by toxemia, or may be very slight until a few hours before death. Similar peritonitis is rarely due to necrosis of a lymph-node, or extension from the wall of the gut.

Cholecystitis is not very rare during typhoid or later (months or years). Typhoid bacilli are often found in the gall-bladder and may live there for years. *Gall-stones* are probably often due to typhoid.

Circulatory: The *blood* shows a progressive secondary anemia, reaching its height at convalescence. The *leukocytes* usually diminish slowly but steadily into convalescence, due chiefly to an absolute decrease in polynuclears; the eosinophiles are decreased during the fever, but there is eosinophilia in convalescence. Complications are usually inflammatory and cause leukocytosis. A cold bath increases the leukocytes temporarily. In most cases typhoid bacilli may be demonstrated in the blood by means of cultures.

The Heart.—Sudden death may occur late in the fever or in convalescence; the heart action grows rapid, weak, irregular, and stops within a few seconds or hours. There may be endocarditis, with typhoid bacilli on the valves, or pericarditis. Thrombosis of arteries, or more often of veins (typhoid bacilli may be found in clot), with phlebitis, especially femoral, may occur late or in convalescence. Symptoms of femoral phlebitis: a few days of higher fever, leukocytosis, local pain, tenderness and induration over the site of the thrombus; later, edema of the leg, which may last many months or even years. Rarely the thrombus is dislodged and causes embolism (lung, etc.), or suppurates and causes pyemia. It may form in the veins of the calf; or in the dural sinuses or cranial veins (causing hemiplegia, aphasia, or other paralyses), or in other veins. Infarcts may form in brain, lung, spleen, kidney, or spleen.

The Respiratory Organs.—Larynx: Catarrh or ulceration, or perichondritis with stenosis may occur. Slight *bronchitis* is common early, and may persist or be very marked. *The Lungs:* Hypostatic congestion and edema are common in the typhoid state.

Pneumonia.—(*a*) Lobular pneumonia may occur as a secondary infection in the typhoid state. (*b*) Lobar pneumonia: (1) "Pneumo-typhoid" begins like a typical lobar pneumonia; instead of defervescing, it becomes a typical typhoid in the second week. There may be typhoid bacilli only in the lung. (2) In the second or third week a secondary pneumonia may appear. Often the only symptoms are physical signs, a rapid, weak heart, rapid, shallow breathing, and frequently death. Abscess or gangrene and hemoptysis are rare. *Pleurisy* occurs early or late, even in convalescence. It is usually purulent; typhoid bacilli may be found in the exudate.

Temperature: The onset may be abrupt, 103° F. on the first or second day, then it is often preceded by a chill. A sudden fall at any time may be due to no discoverable cause, to hemorrhage or perforation. There may be marked remissions throughout; remissions of 3 or 4 degrees F. are common in the last week. It may fall abruptly at any time and stay down, or afterward rise and run the usual course. In the morning temperature may touch normal and the evening temperature be 104°–105° F. There may be a very irregular temperature throughout or inverse temperature. Afebrile cases have been reported. Complicating inflammations may cause a rise of temperature of 1 to 2 degrees. There may be evening temperature of 100°–101° F. for several days of convalescence. Temperature varies with the severity of the other symptoms in the ordinary case.

Nervous symptoms: "Meningo-typhoid" begins like meningitis, with headache, delirium, and rigidity of neck; after a few days it assumes the typical course of typhoid. A real meningitis is extremely rare. Insanity may be a sequel, but recovery usually follows. Coma vigil may be present. Local or multiple neuritis appears late or in convalescence; is usually in the legs and involves the extensors. "Tender toes" are probably due to a neuritis. Symptoms of a neuritis: Marked tenderness of the nerve, with intense pain along its distribution, and later a loss of function. Usually recovery is quite complete.

Special Senses.—Any part of the eye or its nerves may be inflamed. Otitis media is not rare.

Kidneys.—Febrile urine is very common. Acute nephritis is rather rare. "Nephro-typhoid" begins like a very intense nephritis; after a few days of prodroma there are fever, pain in the back, scanty bloody albuminous urine with many casts; later typical symptoms of typhoid appear. Late or during convalescence nephritis rarely appears. Typhoid bacilli in the urine are common in the second week, and may persist for years without causing discomfort. Cystitis late in the disease is not rare; it continues into convalescence. Pyelitis late or in convalescence is rare.

Orchitis, oöphoritis, and mastitis are rare.

Periostitis may develop late in convalescence or after months or years. Often the pus contains typhoid bacilli only. Usually it is seen in the legs or ribs. There may be necrosis of bone. Periostitis is rather common, and is quite characteristic.

Typhoid spine appears several weeks after invasion. It is usually a neurosis, but sometimes a perispondylitis of the lumbar or sacral region. It is afebrile. There are pain and tenderness along spine. The back is kept rigid and the patient will not or can not get up. They recover in time.

The Joints.—Arthritis is rare and late.

Muscles.—Myositis rarely occurs, with hemorrhage into the muscles. During convalescence abscesses may develop.

Varieties.—*Mild.*—The invasion, free from marked symptoms, is followed by defervescence, the fever lasting only ten to fourteen days. Spots and a large spleen are present; also Widal's reaction, and perhaps bacilli in the blood. *Abortive:* The invasion is rather abrupt, spots appear in two to five days. After seven to ten days there is practically a crisis. *Severe forms* are sometimes called gastro-intestinal, pneumonic, pleuritic, cerebrospinal, or nephritic, with corresponding symptoms very prominent. *Protracted forms:* The symptoms of the second or third week continue for from one to three or more weeks longer; then the patient may defervesce. The typhoid state may be unusually severe as well as protracted. *Ambulatory form:* Symptoms of invasion are milder, and the patient keeps up. The first marked symptoms may be the rash or hemorrhage or perforation or delirium, and the temperature when taken is found to be 103° F. or more. This is a severe form. *Hemorrhagic:* A petechial rash is rare. It runs through the gamut of colors and fades in seven to ten days. General bleeding is very rare. An *afebrile form* has been reported with the temperature never above 100 degrees. In *children* typhoid is rare under two years, but rather common after five. The rash is inconstant, intestinal lesions and symptoms are apt to be mild; early bronchitis is marked; the invasion more abrupt; the nervous symptoms are marked, and may suggest meningitis. The course is short and mild, and the mortality low. In the *aged* the mortality is high; the temperature usually is not high; complications are common, especially heart failure and pneumonia. In *pregnancy* typhoid often is severe, and abortion is usual.

Relapse occurs in from 5 to 10 per cent. (1) After a normal temperature for three days to three weeks there is typical invasion, followed by a large spleen and spots, and bacilli in the blood. The course is mild and averages two weeks. There may be two, three, or even five relapses.

(2) Intercurrent relapse or recrudescence. The temperature stays below 101° F. for two or three days, then rises and resumes a typical course. This is not a true relapse.

Convalescence may be interrupted by: (A) Post-typhoid septicemia. (*a*) Temperature 100°–101° F. for several days. (*b*) Chills, fever (103° F.), and sweats in irregular paroxysms for a week or more. (B) Pyemia with boils or skin abscesses (often cold abscesses); or widely diffused abscesses. (C) A "spurious relapse" (not a relapse or recrudescence). There is an abrupt rise of temperature to 102°–103° F., that lasts three or four days, without leukocytosis. It may be due to food, constipation, excitement, or perhaps to getting up too soon. (D) A rise may be due to any inflammatory complication. (E) A subnormal temperature in convalescence (97° F.) for several days is common. (F) Bradycardia is common. (G) It may be months before the mental and physical strength returns perfectly.

Prognosis and Mortality.—The death-rate varies from 5 to 20 per cent. With tubbing it averages in hospitals 7 or 8 per cent. The prognosis is better in children, in certain epidemics, and with low temperature. It is graver with complications at onset (see "grave cases" above), or with any of the following symptoms unusually marked: Persistent vomiting, diarrhea, tympanites, pulse 130 continuously, arrhythmia, severe nervous symptoms in the typhoid state; also in protracted cases or with complications that are themselves serious. Sudden death is seen late in the fever or in convalescence; the heart becomes rapid, weak, irregular, and stops after a few seconds or hours. Some cases begin mildly and later become severe. The prognosis is bad in the aged and in fat persons. It is worse in women than in men.

Specific Diagnosis.—Isolation of typhoid bacilli from the blood in the second week, rarely in the first week; from the stools and urine in the second week; or from the rose spots; and Widal's reaction, sometimes in the first week, are valuable. Ehrlich's diazo reaction in the second and third weeks is less valuable.

Treatment.—**Prophylactic.**—Typhoid in cities has been much lessened by filtration of the water, the use of water from a pure source, and by the proper collection and disposal of sewage. During an epidemic, boil all water used for drinking, cooking, or washing food, dishes, or the hands and face. Raw foods should be dispensed with if possible. Preventive inoculation may be used. In the sick-room, disinfect all vomitus, stools, urine, sputum, utensils, bedding, and everything that comes into contact with the patient from the time that typhoid is suspected until the patient is well. For the methods see p. 20. Isolation is not customary, although some recommend it.

General Management.—As in all fevers. The patient is kept in bed. The diet is liquid.

Serum Treatment.—Several serums have been tried, but none is in general use.

Antiseptic treatment is not much in favor. Calomel, β-naphtol, chlorin, salol, other salicylic acid compounds, carbolic acid, and thymol have their enthusiastic advocates.

Hydrotherapy.—Tub baths. A full-length tub at the bedside with water enough to cover the patient up to the neck is necessary. The water is renewed daily. The duration of immersion is ten to twenty minutes. The patient is lifted from the bed into the tub, is rubbed gently all over during the bath; frequent cold affusion to head or cold compresses are used. Then he is lifted back to bed, is dried and covered with a blanket. The rectal temperature is taken one-half hour after the bath. *Temperature of water:* The first bath may be at 85° F., the next at 80° F., the next at 75° F., and subsequent baths at 70° F. The water may be at 85° F. at the beginning of every bath, and be cooled during the bath to 70° or 65° F. For a child the temperature is 90°–80° F. The duration of the bath may be increased or lessened. For a weak heart whisky (½ ounce) may be given, from thirty to fifteen minutes before the bath; or a hot drink (water, milk, or whisky) may be given just after the bath. *Indications:* A bath should be given every three hours if the rectal temperature is 103 degrees, or even lower if nervous symptoms are marked. *Contraindications:* Severe abdominal pain, intestinal hemorrhage, perforation, peritonitis, phlebitis, a tendency to collapse in the bath, nephritis, or a state of extreme prostration. *Advantages:* The skin is cleaner, with less tendency to bed-sores; the heart is slowed and strengthened, the breathing is deeper, and hypostatic congestion lessened. The temperature is lowered 1° to 3° F. temporarily, and probably its whole course is lower; the nervous symptoms are mitigated; there are less stupor, delirium, tremor, and subsultus tendinum. Sleep for an hour or two often follows the bath. Above all, the mortality is much less. If tubbing is impossible, cold packs, bed baths, or sponging with iced alcohol may be used. Ice-water enemata have been used.

Symptomatic.—Skin abscesses need surgical treatment. For *vomiting*, lessen the food, or change it, or add cerium oxalate, or sodium bicarbonate, or peptonize the milk. For *diarrhea*, if there are five or more large stools a day, or if there are curds in the stools, modify the milk or stop it. Give bismuth, gr. xx or more every few hours, or after every stool. Opium, camphor, or salol may be added. The colon may be irrigated twice a day. For *constipation*, an enema every day or two days. For *tympanites*, turpentine stupes, turpentine or a congener by mouth, gr. x every 3–4 hours. A rectal tube may be passed from twelve to eighteen inches into the colon and left there one-half hour. For *hemorrhage*, stop the baths, give opium in liberal dose; an ice-bag may be put on the lower right quadrant of the abdomen. The greatest possible quiet for two days after bleeding has stopped is necessary. *Perforation:* Surgical treatment as soon as possible. *Peritonitis:* Possibly surgery may help. For the *heart*, whisky or brandy (4 or more ounces a day), strychnin, or other heart stimulant may be needed. *Thrombosis and phlebitis:* Stop the baths, elevate and give absolute rest to the limb when possible. Later, for the local edema use elastic bandages and massage. *Nervous symptoms:* Tubbing relieves most of the worst symptoms. Stupor needs stimulation with alcohol and strychnin, and feeding at regular intervals. *Bacilli in urine:* Urotropin or similar drug, gr. vij–x t. i. d. for a week, usually cures this condition. *Complications* are treated as usual. *Relapses* are treated like the original illness.

Convalescence.—Feeding.—Authorities conflict here. Some give solid food on the first day after the temperature stays normal if the patient is very hungry; others not for ten days. All begin very cautiously with small amounts of plain food, and increase gradually. The patient may sit up after a week or ten days if he feels strong and well. Activity should be increased gradually.

QUESTIONS.

Give the etiology of typhoid.
Give the lesions in the intestine, lymph-nodes, and spleen.

What are the incubation period and the prodromal symptoms?

Give the symptoms of an uncomplicated four weeks' typhoid by weeks.

What are the principal complications?

Give the symptoms of intestinal hemorrhage, of perforation, of general peritonitis, of pneumonia.

What is pneumo-typhoid? Nephro-typhoid?

How may the temperature vary from the typical course?

Draw the typical temperature curve.

Give the chief variations from the typical four weeks' course.

What constitutes a relapse?

What are the other causes of fever in convalescence?

What are the specific diagnostic points, and how soon are they present?

Give the preventive treatment as applied to the patient and to the community.

Give the indications, contraindications, advantages, and technic of tub-baths?

Discuss the diet from the onset to the end of convalescence.

TYPHUS FEVER.

Etiology.—The specific cause is not known. Outside the body it seems to lodge in clothing, furniture, and other fomites, retaining its virulence for an indefinite time. It seems to be killed by a temperature of 100° C. and by the ordinary disinfectants. An abundance of fresh air is its greatest foe. It is given off in the breath, epidermal scales, and possibly in the stools. It floats in the air and enters the body with the breath. *Predisposing factors:* Typhus occurs at all ages and in both sexes. It is a disease of overcrowding, bad air, and squalor, and therefore is more common among the poor, in unsanitary dwellings, barracks, jails, hospitals, and in cold weather when ventilation becomes worse. It is endemic in parts of Ireland, Russia, and southeastern Europe. From here it spreads with travel in epidemics that used to be very extensive and fatal. It is extremely contagious in bad air, vastly less so in pure air. Second attacks are very rare.

Lesions.—Degeneration of cells in all glands and muscle. The spleen is large and soft; the blood liquid and very dark. The eruption is present.

Symptoms.—Incubation, twelve days. Prodroma are not usual. *Invasion* is very abrupt, with a chill, rapid rise of temperature (103° F. on second day), and other febrile symptoms. Prostration is marked. The face is flushed and dusky, the expression heavy. Active delirium is often present on the second night. The spleen can be felt in this stage, which lasts from three to five days.

Eruptive Stage.—The eruption, a small macule or papule, like a rose spot, appears; after a day or two it becomes petechial, and therefore takes a week to fade. There may be very many spots and the skin has a mottled look. Appearing first on the chest, the rash spreads everywhere except to the face. It appears on the third to the fifth day, reaches its height in two days, and lasts about ten days in all.

Other Symptoms.—At the end of the first week the typhoid state has developed; the temperature remains high for two weeks and then falls by crisis. Convalescence is rather prompt, and after a month the patient is usually quite well. In a fatal case he dies of failure of the heart, in coma, with hyperpyrexia, or of some complication, especially pneumonia.

The **complications** are the usual ones in fevers.

Varieties.—*Mild.*—The rash may not be petechial, and crisis occurs at the end of the first week. In *very intense cases* the patient may die in three or four days, even before the rash appears. In *hemorrhagic cases* there is bleeding into many organs and death follows.

Prognosis.—The mortality varies from 10 to 20 per cent. or more in different epidemics. It is mild in children and progressively more fatal after thirty years. It is very fatal in those previously run down, and in badly ventilated, dirty places.

The *differential diagnosis* from typhoid is based on the history, the temperature curve at the beginning, the defervescence, the intestinal symptoms, the time of development of the typhoid state, the time and character of the rash, and the specific diagnostic points in typhoid.

Treatment.—*Preventive.*—Rigid isolation, with abundance of fresh air, in tents or in a room with windows constantly wide open, summer or winter. The discharges are disinfected. The general management and other treatment are those of all similar fevers.

QUESTIONS.

Give the etiology, duration of incubation, temperature curve, termination, and prognosis.

Describe the rash.

Give the differential diagnosis from typhoid.

Give the preventive treatment.

VARIOLA (Small-pox).

Etiology.—No specific cause has been demonstrated. Outside the body it seems to float in the air, lodge in fomites, and retain its virulence for months, but seems to be killed by the usual disinfectants. In the patient it occurs in the rash, the desquamated crusts and scales, and perhaps in the mucous membranes, the breath, and in discharges. Variola is transmissible in the incubation period and until desquamation ceases. Infection is by inhalation or rarely by inoculation.

Predisposing Factors.—Susceptibilty seems almost universal, but is more marked in infancy, in negroes, and in races not previously exposed. Small-pox may be congenital; it is endemic in many countries and spreads in epidemics. Second attacks are rare. Vaccination confers immunity.

Lesions.—The rash is due to an exudative inflammation going on to suppuration and necrosis (p. 17). The site is the rete of the skin; the vesicle is caused by excessive exudation of serum; the umbilication because the vesicle has not a single cavity, but is subdivided by trabeculæ of cells or fibrin. The necrosis may be limited to the epidermis, and no scarring follows; or it may extend to deeper tissue, and leave scars that may be permanent and depressed. In the mucous membranes the superficial tissue is macerated and ulcers form earlier. In both skin and mucous membrane there is in the surrounding tissue a zone of exudative inflammation of varying extent. It may be very extensive. There may be secondary enlargement of lymph-nodes. The spleen is enlarged, and there are degenerations in the cells of various viscera.

Symptoms.—*Incubation*, twelve days (one to three weeks). Prodroma are usually absent. *Invasion:* A sudden intense fever (p. 22), severe frontal headache, tearing lumbar pains, chill, marked prostration, temperature 103°–105° F. on first day, and continuing high with moderate morning remissions until the third night. Delirium may develop on the first day. In some cases on the second or third day erythematous points like scarlatina or blotches like measles, or even petechiæ, appear usually below the navel on the abdomen or on the inner sides of the thighs. These fade within a day or two. The duration of this stage is three days. The temperature then falls suddenly nearly or quite to normal, and all symptoms disappear or become very slight.

Eruptive Stage.—The Eruption: First, a small red macule; on the next day a papule with shotty feel; at the apex of this a clear vesicle appears on the third or fourth day of the rash. Its contents are clear, and only a part escapes if the vesicle is punctured. The vesicle soon becomes umbilicated—that is, there is a little depression in the middle. The contents then become turbid, and on the sixth day of the eruption, the ninth of the disease, a rounded pustule, not umbilicated, is seen. Two or three days later (the eighth or ninth day of the rash) the pustule usually ruptures and the pus dries to a dirty, yellowish crust. This crust drops off about the fifteenth or sixteenth day of the rash. Some pustules do not rupture, but dry up, and later this dried crust is cast off. There is left a red spot, which may be depressed; it fades to white in a few weeks. It may leave a permanent pitted scar; the spots increase in size up to the fully formed pustule, which is about one-quarter of an inch in diameter. The "halo of the pustule" is a red and indurated zone that always appears with the vesicle and gradually spreads. It fades when the crusts form. There is a fine desquamation of the epithelial scales for about two weeks after the crusts fall off. In the mucous membranes of the mouth and throat the rash is present, but the maceration of the tissue leads to the early formation of ulcers, the stages are less definite, and the secondary inflammation is apt to be more marked and more general. *Site:* Appearing first on forehead, face, scalp, back of hands, and wrists, within a day the rash spreads over the whole body, including the palms and soles. The rash on the mucous membranes may appear a day before that on the skin. There is but one crop. *Varieties of rash:* The spots may be few and far apart; the pustules may be poorly developed and dry more quickly. They are regularly more numerous on the face. If the halos run together, there is diffuse swelling of the skin. If the pustules do not run together, the case is "discrete small-pox." If the pustules do run together where the rash is thickest, the case is one of "confluent small-pox." In this case there may be extensive areas of necrosis of the skin. There may be bleeding into the rash, usually in the pustular stage. *Secondary fever:* The other symptoms vary with the extent of the inflammation of the skin, and are those of burning, itching, and pain in the skin, and of fever (p. 23). The temperature zigzags upward with the inflammation, reaching its height with pustulation (the ninth day of the disease). In the moderate cases lysis then begins, and takes about a week. In severer cases the temperature keeps up for from three days to a week longer, and then lysis follows, if the patient recovers. The maximum temperature may be from 103° to 104° F. in cases that recover. In fatal cases there is often an ante-mortem rise to 107° F. or even more. In the pustular stage there may be the typhoid state. The *blood* shows a leukocytosis throughout. Convalescence is rapid after mild cases, but slow after severe cases.

Complications.—Any complications of fevers may be present. The secondary inflammation around the rash on the mucous membranes brings into prominence glossitis with dysphagia, colitis or proctitis with diarrhea, bronchitis, laryngitis, edema of glottis. There may be abscesses and cellulitis of the skin and subcutaneous tissue. Besides these the more common are pneumonia, hypostatic congestion, and heart failure.

Varieties.—*Modified small-pox* in the vaccinated (varioloid) has a stage of invasion much the same, or milder in every way. The eruption usually consists of a few spots that may not form distinct pustules or leave scars. Secondary fever may be hardly noticeable. Complications or death are rare.

Discrete small-pox is of moderate severity, secondary fever is distinct. Recovery is the rule.

Confluent Small-pox.—The whole course may be hastened, with a rash on the third morning; a temperature that stays above 100° F.; a marked secondary fever (104°–106° F.); the typhoid state and death from sepsis from the skin or from complications. With recovery the duration of the secondary fever is three or four weeks.

Hemorrhagic Small-pox.—Two types: the latin names suggest the symptoms. (1) *Purpura variolosa* has a very severe onset; bleeding appears on the third day in the skin and mucous membranes of the mouth, stomach, colon, respiratory tract, kidneys, and in other organs. The temperature is very high. The patients die on the third to sixth day, and the typical rash may not develop. (2) *Variola pustulosa hæmorrhagica* is like severe typical small-pox up to the vesicular or pustular stage, then the rash becomes hemorrhagic, and death follows in two or three days.

Prognosis varies with the epidemic and the severity of the case. In the vaccinated the mortality is very low, and bears an inverse ratio to the number of vaccinations and the nearness of the time of vaccination. In the unvaccinated small-pox is much graver, being very fatal under one year, bad under five years, better up to fifteen or twenty, and progressively worse afterward. Previous ill health or disease of the heart, kidney, or lungs, extensive rash and deep suppuration, cellulitis of the skin, edema of the larynx, pneumonia, typhoid state early, coma, convulsions, and a petechial initial rash are grave symptoms. With discrete small-pox some die; with confluent, most die; with hemorrhagic, practically all die. The general mortality is from 15 to 35 per cent. The causes of death are toxemia in the initial stage or during pustulation, heart failure, exhaustion or some complication.

Sequels.—Pock-marks, blindness, otitis media, neuritis.

Treatment.—*Prophylactic:* Vaccination regularly and after every exposure. Rigid isolation of all cases far from other dwellings. Much fresh air. Disinfect all discharges, the skin, and everything that has come near the patient, also the room and its contents after the patient has left it. Continue isolation until the skin is perfectly free from scales.

For general management and symptomatic treatment, see p. 25.

For the *eruption*, wet compresses give great comfort. For the *crusts*, glycerin or petrolatum is used. The eyes should be carefully washed every few hours with a mild antiseptic. Special care of mouth is needed. Hydrotherapy, as in typhoid, is indicated for high temperature or nervous symptoms.

QUESTIONS.

Give the etiology, the duration of the incubation period, the symptoms of the periods of invasion, and of eruption.

Describe the initial rashes.

Describe the characteristic rash.

Draw the temperature curve of a typical case.

What are varioloid, discrete small-pox, confluent small-pox, hemorrhagic small-pox?

Discuss the prognosis.

What is the preventive treatment?

VACCINIA AND VACCINATION.

Cow-pox is a disease of cattle, transmissible to man by inoculation; it is generally thought to be modified small-pox. *The lymph:* The purulent matter from the pustules on the cow is mixed with glycerin and kept for a few weeks before it is ready for use. Instead of glycerinated lymph, dry points may be prepared by dipping a piece of ivory, bone, or quill in the pus and letting it dry. The purulent contents of the vesicle on the eighth day in a child may be used in exactly the same way. *The operation:* The skin near the deltoid insertion or on the leg is cleaned and dried. It is scratched with a needle or scraped with a knife until serum exudes. Then the virus is rubbed in and allowed to dry. The spot may be covered with sterile gauze. *Symptoms:* At first the scratches heal like any other scratches; this is the incubation stage, and lasts about two days. Then the eruptive stage begins. First a papule, then a vesicle, umbilicated, which increases in size until the eighth day. During the second week the contents become purulent, escape, dry, and form a crust. At the end of the third week the crusts fall off, leaving a red and pitted scar. Later it fades to white, but the pits persist. Around the vesicle is a zone of exudative inflammation that increases in the pustular stage, and may be two inches wide; it resolves when the crusts form. The related lymph-nodes are inflamed, enlarged, and tender. *Other symptoms:* The lesion itches, and is painful and tender during active inflammation. There are often a fever varying with the inflammation, and a leukocytosis.

Variations.—The pock may develop faster or slower, there may be a group of little pocks around it, or there may be a more or less general erythematous, papular, or pustular rash. Secondary vaccination may be quite like a primary, but usually the pock is smaller, the secondary inflammation less, and the scar smaller and less distinct.

Complications.—Septic infection, erysipelas, tetanus, syphilis. Some add tuberculosis and leprosy.

Vaccination protects pretty thoroughly for a few years. The more numerous the successful vaccinations the greater the protection; with four good scars it is almost complete. Vaccinate a child at about three months, again at about ten or twelve years, and thereafter every seven years, during prevalence of the disease and after every exposure. If the pock is not quite typical, revaccinate at once. An unsuccessful vaccination does not necessarily mean that the patient is immune.

QUESTIONS.

What is the source of vaccine?
How should you vaccinate? When? How often?
Describe the lesion after a successful vaccination.
What general symptoms are common?
Discuss the immunity conferred.

VARICELLA (Chicken-pox).

Etiology.—The specific cause is not known, but lives for a time on fomites, and is in the vesicle and crusts. It is transmissible until all crusts have disappeared. *Predisposing factors:* The most common age is from three to six years. Epidemics occur in schools, etc. Sporadic cases are seen. Second attacks are rare. The lesion is the rash.

Symptoms.—Incubation, two weeks. Stage of invasion may be unnoticed or there may be slight fever for a part of a day. *Eruption:* A red macule or papule appears; in a few hours it becomes a hemispheric, monolocular vesicle rising out of normal or slightly reddened skin, and containing clear liquid. Sometimes it is umbilicated. On the third or fourth day the contents are turbid; on the fifth or sixth it has dried to a brown crust. This falls off after a few days. Scratching or dirtiness may result in suppuration, with deep enough necrosis to leave a permanent, depressed scar. This is more common in sickly children. The rash appears on the first day, on the trunk, often on the scalp, sometimes on the face or elsewhere. A modified rash may appear in the mouth and throat. New crops appear before the earlier spots have faded. The total duration of the rash is from one to three weeks. *General symptoms:* A little fever for the first three or four days is sometimes present.

The **prognosis** is good.

The child should be isolated until the crusts disappear.

QUESTIONS.

What is the incubation period of varicella?
Describe the rash.
Differentiate a marked varicella from a rather mild variola.

SCARLET FEVER (Scarlatina).

Etiology.—The specific cause is not known. Outside the body it lives for a long time (years) in fomites; it may float in the air. In the body it occurs in blood, tears, secretions of nose, throat, and bronchi, and in the scales of the skin. It is directly contagious from the onset to the end of desquamation. It probably enters the body with the breath. *Predisposing factors:* Age, usually two to ten years. Most cases occur in the fall, when schools are in session. Previous ill health may increase susceptibility. Epidemics are common. It is endemic in towns, and sporadic cases occur. Natural immunity is not very common, but second attacks are rare.

Symptoms.—Incubation, two to four days. *Invasion:* An abrupt fever with sore throat, vomiting, and headache. This stage lasts one day. *Eruptive stage:* Rash. Two elements: (1) Diffuse erythema that comes out after and goes before the (2) fine red papules, which remain bright from a few hours to three days and then fade. On mucous membranes of the mouth there is diffuse congestion with some redder points (see also below). The rash appears on the second day, first on the sides of the neck and on the chest; spreads within a day to the face, and perhaps the entire body except the lips and chin. There is one crop which lasts four or five days. Desquamation in large flakes follows, and continues from one to three weeks. *Varieties of rash:* There may be very few spots with little or no diffuse erythema. A little vesicle may surmount the papule. There may be petechial spots, or, if the rash is copious, some swelling of the skin. *Other symptoms:* There is a general redness of the mouth, tonsils, and pharynx, with redder points. There may be a false membrane on the tonsils. The tongue at first is white and coated; after a day or two the fungiform papillæ redden and swell, forming the strawberry tongue. In severer cases the whole tongue may look red and raw with swollen papillæ. There may be a nasal discharge. There are pain on swallowing or speaking, nasal obstruction and deafness or tinnitus. There may be large lymph-nodes or diffuse induration in the neck, with pain and tenderness. The temperature may reach 103° F. on the first day, it stays high for one or two days, and then falls by slow lysis, lasting to the eighth or ninth day. A leukocytosis is present.

Complications.—Acute endocarditis at any stage; pleurisy with effusion, especially in convalescence; pneumonia in severe cases; edema of the glottis; suppuration of lymph-nodes in the neck; purulent otitis media. Nephritis is common from the second to the fourth week, even after mild cases. Arthritis may develop late or in convalescence, with symptoms like those of acute rheumatism. Real diphtheria sometimes follows or accompanies scarlatina. *Sequels:* Chronic endocarditis, chronic otitis media, chronic nephritis, chronic enlargement of cervical nodes.

Varieties.—*Abortive* cases (scarlatina sine eruptione) have only a sore throat and fever, but may give rise to a severe case in another patient. The rash is fugacious or absent. *Malignant forms:* (1) Atactic, with delirium, hyperpyrexia, coma, and death in one or two days. (2) Anginose form. There are marked swelling of the throat, toxemia, and death in three or four days from suffocation or poisoning. Or the patient may have extensive necrosis of the tissues with ulcer formation, and a fetid discharge from the nose, mouth, and ears, secondary pneumonia, and death. (3) Hemorrhagic cases. Bleeding into the skin, mucous membranes, and kidneys, with toxic symptoms and death in two or three days.

Prognosis.—In previously healthy children in good homes death is rare, but the sequels may ensue. In hospitals the mortality is about 10 per cent., or in some epidemics higher. Under one year scarlatina is very fatal; most deaths are before six years.

Treatment.—Prophylactic. Isolation should be strict until desquamation ceases. Disinfect the discharges from nose, mouth, and ears, and the desquamated epithelium. The skin is anointed to prevent dissemination of the scales. General treatment as in other fevers. The care of the mouth and throat is most important. Hydrotherapy is useful. The patient may get up after a week of convalescence.

QUESTIONS.

Give the etiology and the duration of the periods of incubation and of invasion of scarlet fever.

Describe the rash.

Draw the temperature curve.

What are the chief complications and clinical varieties?

Discuss the prognosis.

What is the preventive treatment (including the duration of isolation)?

MEASLES (Rubeola).

Etiology.—The specific cause is not known. Outside the body it lives on fomites, but is killed by ordinary antiseptics. It enters the body with the breath or by inoculation. In the body it is in the secretions of the conjunctival and respiratory mucous membranes, and perhaps in the desquamated epithelium. It is extremely contagious, especially in the earlier stages. *Predisposing factors:* It is most common in the first decade. In new races all ages are very susceptible. It is endemic in towns, and occurs in epidemics, especially where children are crowded together. More cases occur in winter. *Immunity:* Second attacks are more frequent than in other exanthemata, but are not common.

Lesions.—Catarrh of the conjunctivæ and air-passages. Fatal cases usually have pneumonia and enlarged bronchial nodes.

Symptoms.—*Incubation*, two weeks. *Prodroma:* The child may be fretful for two or three days. *Invasion:* Fever, rather abrupt, with red and burning eyes, sneezing, a thin nasal discharge, and cough with little mucous sputum. *Temperature:* The first day, 102°–104° F.; the next two days it remits 1 or 2 degrees. *Koplik's spots* are small red papules with a bluish dot at the apex that appear on the mucous membrane of the cheeks two or three days before the rash. They are pathognomonic of measles. This stage lasts three or four days. *Eruptive stage:* The rash consists of red elevated spots that tend to coalesce into more or less crescentic blotches giving a mottled appearance. They appear on the fourth day on the face and neck, spread in a day to the trunk and limbs, and may be nearly universal. They remain bright two or three days and fade in two or three days. A fine branny desquamation follows and lasts a week. In even moderate cases there may be small vesicles or petechiæ in the rash. Catarrh of the mucous membranes and Koplik's spots last until the rash is fading.

Other Symptoms.—Those of the fever and catarrh are present. The lymph-nodes of the neck are often swollen and tender. The fever is highest on the day the rash appears, stays quite high until the rash is about to fade at the end of the first week, and then falls by crisis, or sometimes by lysis. There is no leukocytosis.

Complications.—Bronchopneumonia, enteritis, colitis, otitis media, noma of the mouth or vulva.

Varieties.—*Mild cases:* There may be no rash and little fever. *Malignant cases:* (1) May show symptoms of marked toxemia and die in a few days with pneumonia, croupous inflammations of the throat, or noma. (2) Hemorrhagic cases have bleeding into the skin and mucous membranes, high fever, and death in a few days.

Prognosis.—Children previously well, recover. In sickly children, especially in institutions and in races not previously exposed, severe and malignant cases with complications and death are common. After measles tuberculosis seems to be common.

Treatment.—*Prophylactic:* Isolation until the end of desquamation. Disinfection of the discharges and of the inflamed mucous membranes and of the scales of the skin. Anoint the skin during desquamation. Especial care of the mouth and throat is necessary. General management as in other fevers.

QUESTIONS.

Give the etiology, and the duration of the incubation period, and of the period of invasion.

Discuss Koplik's spots.

Describe the rash.

Draw the temperature curve.

What are the common complications and clinical varieties?

Discuss the prognosis.

Give the differential diagnosis from scarlatina, from typhus, and from variola on the day the rashes appear, and also later.

RUBELLA (German Measles).

Etiology.—The specific cause is not known. It is transmitted usually by direct contact, and possibly by fomites. It is contagious in the last part of incubation, and probably to the end of desquamation. It is usually seen from the fifth to the fifteenth year. Epidemics usually occur in the winter and spring. Second attacks are rare.

Symptoms.—*Incubation:* Two weeks, more or less. *Invasion:* Often there are no symptoms until the rash appears, but there may be slight fever (100° F.) and slight catarrh of conjunctiva, nose, throat, and bronchi. The lymph-nodes behind the ears and at the angle of the jaw are enlarged and somewhat tender. This stage may last one or two days. *Eruption:* Two types: (1) Papules appear one-fourth inch in diameter, somewhat raised, red, fading temporarily on pressure, but do not form big blotches. (2) There may be a general redness due to many fine red points. Both forms may be present in the same patient. The rash appears as the first symptom or after one or two days of slight illness, behind the ears, around the mouth, and later on the chest or elsewhere. It lasts two or three days. There may be a fine desquamation for a week, varying with the amount of rash. The mucous membranes show only a catarrhal inflammation and swollen tonsils. The slight fever and catarrhal symptoms may last a day or two after the rash appears. Convalescence is very prompt.

The **prognosis** is good.

Treatment.—Moderate isolation.

QUESTIONS.

Give the etiology and incubation period of German measles.

Describe the rash.

Give the differential diagnosis from measles and scarlatina on the day the rashes appear, and also later.

DENGUE (Break-bone or Dandy Fever).

Etiology.—The specific cause is unknown. It occurs in epidemics attacking almost everyone exposed; in summer; in subtropical or tropical climates; spreads rapidly and may exhaust the soil in two months. Second attacks are not common. Lesions are not known.

Symptoms.—*Incubation*, one to four days. The course is divided into *initial fever*, *remission*, and *secondary fever*. *Initial stage:* The onset is abrupt. The temperature rises rapidly to 103° F. on first day; later it may reach 105° or 106° F. In the bones, muscles, joints, and especially in the head and eyes, are intense aching and tearing pains. There is acute arthritis, with pain, tenderness, swelling, and redness. The initial rash is a marked redness of the skin and the visible mucous membranes due to congestion. This may be associated with burning and itching. The prostration is marked. On the third or fourth day a crisis occurs, and the patient feels well but very stiff and somewhat sore. The *remission* lasts two to four days; then there is a similar attack, but milder (*secondary fever*). The secondary rash, however, is different. There are red papules, one-eighth to one-half inch in diameter, that come first on the hands and wrists, and soon spread to the whole body. The spots may coalesce to form large blotches. They fade after a day or two. Desquamation, fine and branny, follows for a week or two. When the rash fades, the febrile and other symptoms subside. The entire course lasts from five to nine days.

Convalescence is apt to be slow because of the sequels, which are enlarged, tender lymph-nodes, especially of the neck, stiffness and pain of the muscles and joints, and muscular and mental depression.

The **prognosis** is good.

Treatment.—Isolation is advisable if general exposure has not already occurred.

QUESTIONS.

Give the etiology of a dengue and the duration of an epidemic in a community.

What are the stages and the chief symptoms of each stage?

ERYSIPELAS.

Etiology.—Streptococcus erysipelatis (erysipelatos, erysipelatosus). It lives for an unknown time in fomites, in the desquamated scales, and in dust, but is killed by the ordinary antiseptics. Within the body it is in the lesion. It enters through the lymph-spaces of an abraded surface or open wound, or possibly, at times, through the intact skin or mucous membranes. *Predisposing factors:* All ages, even the newborn. It is more common in the spring; occurs in epidemics; is endemic in buildings, such as dirty hospitals; and sporadic cases also occur. An attack seems to give immunity for two or three months, then the individual may be unusually susceptible. Repeated attacks are not rare.

Lesions.—An exudative inflammation of the skin or mucous membrane (see p. 17), with much edema in the soft tissues. Colonies of the bacterium abound in the advancing zone of inflammation. There may be bullæ with clear or turbid contents. The lymph-nodes are inflamed.

Symptoms.—*Incubation,* variously given from three to fourteen days. In inoculation experiments in men it was from fifteen to sixty-one hours. *Onset* is that of an abrupt fever (p. 22). *Local symptoms:* At first a small area is red, swollen, and burning. In facial erysipelas this is usually on the bridge of the nose or near the mouth. The inflammation spreads radially; at its edge there is a sharp, wavy ridge of induration with lines of deep induration radiating beyond this. The edema causes great swelling, especially in loose tissue like the eyelids. In a given spot the inflammation resolves after about four days, and the lesion then may be somewhat annular. There may be blebs in the epidermis, with clear or cloudy contents, which escape and form a yellow crust. In the mucous membranes the lesion is similar and the edema marked; it may appear in the nose, mouth, throat, larynx, or vulva. The lymph-nodes are enlarged and tender. The inflammation may spread irregularly over a large part of the body and last several weeks (wandering erysipelas). A fine, branny desquamation follows. *General symptoms* are those of fever (p. 23). There is a rapid rise to 103°–105° F. on the first day. The maximum, 1 or 2 degrees higher, is reached on the second or third day. Daily remissions of 2 to 4 degrees occur. Leukocytosis is present throughout. When the active inflammation in the skin stops there is a crisis; this is usually at the end of a week in the moderate cases in previously healthy persons.

Variations and Complications.—Mild cases have very little general disturbance or fever. In those previously weak and in surgical and puerperal cases severer inflammations are apt to occur. Abscesses, or gangrene of the skin or mucous membranes, or edema of the glottis with suffocation, otitis media, pneumonia, malignant endocarditis, an arthritis (often called rheumatism), or pyemia may be present. In the severer cases the typhoid state may develop. In the newborn infection at the navel is usually fatal. In older babies erysipelas may be followed by marasmus.

Prognosis.—The mortality is 4 to 5 per cent. It is more fatal under five or six years.

Sequels.—Temporary loss of hair in scalp cases; abscesses of the skin, especially of the eyelids; otitis media. Relapses are common.

Treatment.—Isolation. Disinfect discharges, surgical dressings, and the scales of the skin. The local treatment is surgical.

QUESTIONS.

Give the etiology of erysipelas.
Describe the lesion.
Describe the general symptoms and the local symptoms.
Discuss the prognosis.
What is the preventive treatment?

LEPTOMENINGITIS.

The different varieties of acute leptomeningitis are discussed together at this point in the hope that the student will find the arrangement convenient.

The General Lesions.—The bones of the cranium are congested. The dura is tense; its inner surface is dry. The pia and arachnoid are the seat of an exudative inflammation (p. 17) with serum, fibrin, red cells, pus, and bacteria in varying proportions and amount. The pus may be pasty and greenish yellow. Edema is often present. The lesions are usually more marked in the sulci. The brain may look flattened. It shows an exudative inflammation most marked along the vessels, with degeneration of the nerve-cells. There may be softening, edema, petechiæ, or minute abscesses. The ependyma is the seat of an exudative inflammation, and the ventricles may be distended with clear or turbid serum or with thin pus. The distention seems to vary with the duration of the inflammation. The cord and its membranes are similarly involved to a varying extent. The inflammation may extend along the sheaths of the nerves and the nerves may degenerate. The inflammation may be confined to a small part of the meninges, cranial or spinal, the vertex, the base, or it may be general. Death may occur at any stage, even during congestion. With recovery the products of exudation are absorbed, but often connective tissue is formed and persists. The nerve-cells may recover or suffer more or less permanent damage. The other cells of the body show the usual degenerations.

EPIDEMIC CEREBROSPINAL MENINGITIS.

Etiology. — Diplococcus intracellularis meningitidis (Weichselbaum). Outside the body it is unknown except in artificial cultures. It may live a short time on fomites and in the air. In the body it occurs in the exudate in the nervous system, in the nasal discharge, and sometimes elsewhere, especially in the blood and joints. The disease may be directly contagious, and germs may get in through the nose. Children and young adults, especially in unsanitary dwellings, are most susceptible. It occurs in temperate climates, in limited epidemics, often confined to a barrack or village, and usually in winter. Sporadic cases occur especially after an epidemic. Little is known of immunity, but second attacks have been reported.

Lesions.—The exudate is usually greatest at the base of the brain, and the posterior surface of the thoracic and lumbar cord. The diplococcus is present in the serum and in the pus-cells. Involvement of the nerves and their sheaths is common.

Symptoms.—*Incubation:* Not known. Prodroma occasionally are present for two or three days; some headache, lassitude, aching back and limbs. The *onset* often is abrupt. A chill (convulsion in the young child) is common. There are intense headache, often occipital, pain and rigidity of the muscles of the neck and back, vomiting, and a temperature of 101°–103° F. The general symptoms of fever are present, but the temperature is irregular (101°–104° F.), and often not very high except shortly before death, when there may be hyperpyrexia. Leukocytosis is present throughout. The clinical picture depends on the nervous symptoms. In general there are at first symptoms of intensified, later of depressed nervous action. There may be an intermediate period with alternating excitation and depression. These symptoms are early, active and marked delirium, fading into stupor, in which the patient may cry out as from pain (hydrocephalic cry), and finally coma. *Special senses:* Early there are vertigo and photophobia, later blindness. Painful hearing, later deafness. Strabismus or nystagmus; pupils contracted, later dilated and irregular. Ptosis. A tense expression of pain; later there may be facial paralysis. The pulse is slow, perhaps irregular; later very fast, irregular, and weak. The tache cerebrale and flushing are present. The breathing is often sighing, but may be Cheyne-Stokes. There are sudden, forcible, or regurgitating vomiting, even when the stomach is empty (an early symptom), and marked constipation; headache is very severe and constant, usually occipital, and evident even in marked stupor. General hyperesthesia is present in the skin, muscles, and bones. The spine is tender and painful. There is tonic spasm of the muscles of the back of the neck and along the spine, and to a less extent in the limbs also. Muscular twitching may occur. Passive motion causes pain and is resisted. The limbs are half-flexed, the head much retracted, the rest of the spine straight or in lordosis but rigid, the abdomen scaphoid. Kernig's sign[1] is present. Even in the late stages, except for a short time before death, it is not common to have anesthesia or paralysis. Sometimes incontinence of feces and urine appears. The reflexes are variable. Emaciation is rapid from the onset. If the patient recovers, the functions gradually become normal. Convalescence is often very slow. Sequels are common, and may last for weeks or months, or be permanent. *Skin symptoms:* Besides the tache cerebrale, which is not confined to meningitis, there is very often herpes of the lips and face, and sometimes elsewhere; also a petechial rash, rose spots, urticaria, bullæ, or ecthyma. These rashes vary in frequency, extent, time of appearance, and duration. They are often absent.

Complications and Sequels.—Bedsores are not rare. There are sometimes enteritis and diarrhea. Rhinitis is common. Pneumonia sometimes occurs with Diplococcus intracellularis only. Headache may persist for months. Chronic hydrocephalus may appear after a few weeks of convalescence. Mental feebleness or aphasia, hemiplegia, paraplegia, or monoplegias may follow. There may be optic neuritis, optic atrophy, or other inflammations of the eye that may cause permanent blindness. There may be permanent deafness, or in very young children deaf-mutism. The senses of smell and taste may be lost. Arthritis, like rheumatism, is a rather common complication (diplococci may be found in effusion). Any other pyemic complication may occur.

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[1] If the thigh is extended the leg can be extended, but if the thigh is flexed to form a right angle with the trunk, the leg cannot be fully extended either actively or passively.

Varieties.—*Mild cases* with slight headache, stiffness and pain in the neck, back, and limbs, and little fever, last two or three days. They are diagnosed only in epidemics. *Abortive cases* start like severe cases, but after two to four days cease abruptly. *Ordinary cases* have remissions and exacerbations for one to three weeks, and convalesce slowly. Half of the deaths occur in the first week. *Intermittent cases* have fever like pyemia, with exacerbations on alternate days and intermissions or marked remissions. *Malignant cases* end in death after twelve hours to three days. There may be extensive bleeding in the skin and elsewhere. *Chronic cases* drag on for two to five months, with exacerbations and remissions or intermissions, and are fatal.

Prognosis.—Worse in young children, after forty, and at the beginning of an epidemic. Under two years almost all die; up to five most die. The mortality varies from 25 to 70 per cent. in different epidemics.

Lumbar puncture: The patient lies on the right side, with thighs flexed. The skin is cleaned and may be frozen. A long, slender aspirating-needle is thrust forward, slightly inward and toward the head into the spinal canal (2–8 cm.), starting 2 to 3 cm. to the right of the middle line, opposite the interval between the third and fourth lumbar vertebræ. The liquid drops out slowly. It is usually turbid, the cells and serum contain Diplococcus intracellularis, which are seen in smears and may be cultivated.

Treatment.—Isolation is not customary, but some advise it. General management as in other fevers. *Special treatment:* Ice to the head and spine is used continuously. Tub baths at 98°–104° F. for ten minutes every three hours may lessen muscular spasm and pain. Morphin hypodermically every four or six hours, or bromids are given. Counterirritation to the nape of neck in early stages is used. Some think that mercury or potassium iodid aids absorption. In coma and convulsions withdrawal of cerebrospinal liquid by lumbar puncture sometimes helps.

QUESTIONS.

Give the general lesions of meningitis.

Give the etiology, the lesions, the general symptoms of the ordinary case, the skin-symptoms, complications and sequels of epidemic cerebrospinal meningitis.

Discuss the prognosis.

What is Kernig's sign? Describe lumbar puncture and its uses.

Give the treatment.

SECONDARY PURULENT MENINGITIS.

This in a general way is like the preceding. The points of difference are:

Etiology.—It is a secondary disease and is due to: (1) Extension from otitis media or interna, mastoiditis, caries of the petrous part of the temporal bone, from the nose or frontal sinuses, the sinuses of the dura, or cerebral abscess. (2) Trauma of the head, sometimes even without fracture. (3) Infectious diseases—pneumonia, malignant endocarditis, erysipelas, rheumatism, pyemia, or less often any other infection. (4) It may be a terminal infection in arteritis, endocarditis, nephritis, gout, or cachexias, especially in children. *Bacteria:* Pus cocci or pneumococcus usually, rarely the typhoid bacillus, colon bacillus, or gonococcus. These may often be demonstrated by cultures from the liquid obtained by lumbar puncture.

Lesions.—There are the bacteria as given above. The site and extent of the inflammation vary much. It may be only on the convexity (pneumonia, malignant endocarditis); at the base (children and terminal forms); unilateral or more limited (otitis, sinus diseases, abscess). The ventricles are seldom much distended. Spinal involvement is less common.

Symptoms.—Those of the primary disease are present. The onset of the meningitis is often insidious. The temperature may rise a little and the patient grows more ill. Meningeal symptoms may be few. Often localizing symptoms appear first. Depression symptoms are early and marked. Persistent retraction of the head is less. Death is usual in a few days to two weeks.

Prognosis.—Recovery is rare.

Treatment is the same as in the epidemic form, but certain cases may be saved by an early operation (otitis, cerebral abscess).

QUESTIONS.

Give the etiology and the lesions of secondary purulent meningitis. How do the symptoms differ from those of epidemic meningitis?

TUBERCULAR MENINGITIS.

Etiology.—Tuberculosis elsewhere in the body, especially in the bronchial or mesenteric nodes. *Age*, usually from six months to five years; less often up to young adult life.

Lesions.—In the *meninges* miliary tubercles (see p. 106) are found along the vessels at the base, in the deep fissures, along the arteries of the perforated spaces and the middle cerebral artery, or they may be quite general. There is an exudative inflammation, more marked at the base, and often purulent. The brain is flattened, soft, anemic, has tubercles along the small blood-vessels, and may be infiltrated with leukocytes. The ependyma shows tubercles. The ventricles, especially in babies, are much distended with serum (acute hydrocephalus). A most important lesion is tubercle elsewhere.

Symptoms.—Prodroma are important and last from two weeks to three months, and are more noticeable in patients in fair health. They include languor; marked irritability of temper and peevishness; speech alterations, such as the use of the wrong word and temporary aphasia; constipation, vomiting, especially at night; perhaps enlargement of the nodes of the neck, and loss of weight. In older persons there may be hysteric symptoms, depression, and headache. Rarely the onset is sudden. The symptoms are divided into three stages: (1) excitement, (2) transitional, and (3) paralysis, with the symptoms given on p. 56. The temperature may range from 101° to 103° F., but for a few days before death there may be a subnormal temperature or hyperpyrexia. The pulse in the first and second stages is more irregular and slow than in other forms of meningitis. Leukocytosis may be absent. The hydrocephalic cry is perhaps more common. Retraction of the head is less constant. The eye may show tubercles in the choroid. There is usually no herpes or rash, but flushing may be present.

The **course** is slower, lasting two to four or five weeks.

Sometimes there is a rapid course, especially in adults. Practically all die.

Lumbar puncture: Tubercle bacilli may often be demonstrated in smears or by animal inoculation, but a sterile liquid suggests tuberculosis. Tuberculin injection has been used for diagnosis.

The **treatment** is like that of other meningitis.

QUESTIONS.

Give the points in the etiology, lesions, and symptoms of tubercular meningitis as it occurs in children, that bear on the differential diagnosis of meningitis.

Give a connected account of all of the symptoms of tubercular meningitis.

MUMPS (Infectious Parotitis).

Etiology.—The specific cause is unknown. It is contagious from the onset until after resolution. It usually occurs in childhood and adolescence, and is more common in boys. Epidemics occur, especially in the spring and fall. It is endemic in most cities. Second attacks in the same gland are rare.

Lesions.—An exudative inflammation of the parotid, terminating in resolution.

Symptoms.—*Incubation:* Usually given as from two to three weeks. *General symptoms:* Moderate fever (101° F.) lasting a week or less. *Parotid symptoms:* Swelling begins, soon after the onset of the general symptoms, in the exact site of the gland. The lobe of the ear is pushed up and out. Later the swelling may be very great. The other parotid is often involved after a day or two. The gland enlarges for three or four days, is stationary for three or four days, and resolves in three or four days. There are ptyalism, aching pain and tension in the face, which are worse during and after motion of the jaw, and with swallowing. Severe cases may have high fever (104° F.), with delirium and marked prostration.

Complications.—Earache, otitis media or interna may occur. Orchitis (after puberty) may appear in convalescence. It usually lasts three or four days, and atrophy of the testis may follow. Vulvovaginitis or mastitis may occur in girls, or very rarely oöphoritis.

The **prognosis** is good.

Treatment.—The general treatment depends upon the severity of the general symptoms. Locally, heat to the face and neck gives relief.

QUESTION.

Give the etiology, incubation period, symptoms, and complications of mumps.

WHOOPING-COUGH (Pertussis).

Etiology.—The specific cause has not been demonstrated. It is probably given off in the sputum and the breath. It enters the body in the breath. It is directly contagious. The contagiousness is more marked in the catarrhal stage, and probably ceases within six weeks. Transmission by fomites is not common. Pertussis is most common in infants and young children, and is apt to follow measles or general ill health. Epidemics usually occur in the spring and fall. It is endemic in cities. Second attacks are not common.

Lesions.—Acute catarrh of the respiratory mucous membranes. Fatal cases have also collapse of the lung or bronchopneumonia and enlarged bronchial nodes.

Symptoms.—*Incubation*, four days to two weeks or more. Two stages: (1) the *catarrhal* stage begins as a moderate bronchitis, with catarrh of the upper air-passages also. There is fever (100°–101° F.) for a few days. The cough grows worse, especially at night, and after a week or two the (2) *paroxysmal stage* develops. The *paroxysm:* There is a series of dry, short coughs, the face gets red and very congested, the eyelids swell, the heart is rapid and weak, and the pulse small; the patient is nervous and terrified. Finally he gives a long inspiratory whoop and the color clears. Vomiting of ordinary stomach contents and much mucus is common. There may be involuntary defecation or urination. A little mucous sputum may appear with the cough. Paroxysms are induced by emotions or dusty air or feeding. Two or three may follow quickly, and the child may sink back exhausted.

There may be from three to fifty a day, occurring more often at night. In time they grow milder and less frequent, and cease after two to ten weeks. The general condition may be very good throughout, or the child may show exhaustion and lose weight. Physical signs suggest a slight bronchitis. An ulcer on the frenum of the tongue is common in the paroxysmal stage. Leukocytosis and lymphocytosis are present.

Complications.—Vomiting and diarrhea may be marked. During a paroxysm there may be bleeding into the eyelids, conjunctivæ, nose, throat, or within the cranium, with consequent convulsions and paralyses. Collapse of the lung occurs in babies. Bronchopneumonia is seen. Convulsions may occur without intracranial lesion. Acute nephritis is rare. Vesicular emphysema is common. Interstitial emphysema is serious but not common.

The **prognosis** is good in a robust child, but many children die with pneumonia or enterocolitis.

Treatment.—Isolate from other children. Disinfect the sputum and vomitus. General management varies with the general condition of patient. *Drugs:* The most popular are belladonna in full dosage, antipyrin, vaporized carbolic acid or its derivatives.

QUESTIONS.

Discuss the etiology of whooping-cough.

What are the incubation period, the symptoms, complications, and sequels?

Discuss the prognosis and the treatment.

How early would you expect to diagnose a case of whooping-cough?

INFLUENZA.

Etiology.—The specific cause is probably Bacillus influenzæ (Pfeiffer). It occurs in discharges from inflamed mucous membranes, is given off in coughing and perhaps in the breath; it enters the body with the breath or in unknown ways. It is directly contagious, and perhaps is transmitted by fomites also. Epidemics encircle the earth (pandemics) along lines of travel. Smaller epidemics occur, and sporadic cases appear for some years after an epidemic. An epidemic lasts six to eight weeks in one place. An attack gives no immunity. Most persons are susceptible.

The **lesions** are acute catarrh of mucous membranes with B. influenzæ in the discharges, and the complicating lesions.

Symptoms.—*Incubation*, two to four days. The chief clinical varieties and the complications occurring with them are as follows: (1) *Respiratory:* Symptoms of acute coryza (often with extension to the conjunctiva, frontal and maxillary sinuses, and ear); catarrh of the throat and bronchi with B. influenzæ in discharges; sometimes bronchopneumonia; lobar pneumonia; pleurisy, usually purulent; or abscess or gangrene of the lung. (2) *Gastro-intestinal:* There are marked nausea, vomiting, diarrhea, and abdominal pain. (3) *Nervous:* There are often headache, delirium, vertigo, mental depression, stupor, insomnia, intense aching of the back, limbs, and joints, and extreme muscular prostration. There may be symptoms of meningitis, or encephalitis with paralyses; of neuritis anywhere (including the cardiac, motor, and sensory nerves). (4) *Febrile form:* The temperature may be much or little, continuous or remittent. There may be chills. Almost any organ may be involved in the disease, and many other groups are described. The symptoms may be limited to a single group, but the mixed forms are more common, in which symptoms from various groups are combined and blended in various ways. Probably the *most common cases* combine the respiratory, nervous, and febrile groups, thus: there are chilly sensations or a chill, with rise of temperature to 102° or 104° F., and with the usual febrile symptoms, of which the headache, pain in eyes, back, and limbs, and the physical prostration are extreme. Also there are sneezing, a watery discharge from the eyes and nose, sore throat, slight cough with little mucous sputum. The fever and pains last two to seven days and gradually disappear; the weakness persists several days longer. The catarrhs run an ordinary course. Leukocytosis is usually absent.

Complications are common, especially pneumonia.

Prognosis.—The mortality is from 0.5 to 2 per cent. Death results from complications, especially pneumonia, and in persons with chronic disease of lungs, heart, or kidney. The prognosis is worse at the extremes of life. Sequels are common, such as chronic gastro-intestinal catarrh, palpitation, tachycardia, arrhythmia, chronic bronchitis, bronchiectasis, insomnia, neuralgias, migraine, mental dulness or depression of spirits, and inflammation of eye or ear. Melancholia may follow.

Treatment.—Isolation is advisable. Disinfect the discharges from the mucous membranes. *General:* Confine the patient to bed until the temperature has been normal for a few days. The other treatment is that of fevers. The drugs most used are quinin, phenacetin, salicylic acid compounds, opium, strychnin, and alcohol. Complications are treated as usual

QUESTIONS.

Give the etiology, incubation period, and the symptoms of the usual cases of epidemic influenza.

What are the clinical varieties, the complications, and sequels.

Discuss the prognosis and mortality.

What is the treatment?

LOBAR PNEUMONIA (Croupous Pneumonia).

Etiology.—The specific cause is Micrococcus or Diplococcus lanceolatus or pneumoniæ (pneumococcus). This bacterium seems to cause many lesions, such as primary or secondary lobar pneumonia and bronchopneumonia, pleurisies, endocarditis, pericarditis, meningitis, synovitis. It may occur in the blood and throughout the tissues in a typical septicemia. It may be found both as virulent and non-virulent forms in secretions of the respiratory and oral mucous membrane in health. Perhaps other organisms sometimes cause lobar pneumonia. The bacteria probably enter with the breath. They are given off in the sputum and in coughing, and are found in the dust of rooms. Sometimes pneumonia is directly contagious. *Predisposing factors: Age:* Susceptibility is marked from two to six years, less to about fifteen, and increases steadily thereafter. Susceptibility is greater in males, with exposure to cold and wet, in alcoholics, in the feeble, the sick, and those in unsanitary surroundings. Pneumonia is more common in the late winter and spring and in temperate climates. It is endemic in certain buildings, and sometimes occurs in epidemics. Sporadic cases occur. Repeated attacks are common. Many serums have been tried, but none is accepted as preventive or curative in man.

Lesions.—The inflammation of the lung is not "croupous," but is an exudative inflammation without necrosis. The lesion involves all or most of one lobe, but may extend to other lobes or the other lung. (1) *Stage of congestion:* The involved lobe is enlarged, darker, and tough; it crepitates and floats; the cut surface is moist and red and exudes blood. The pleura is covered with fibrin. Microscopically there is congestion; the air-cells and small bronchi contain some red and white blood-cells, fibrin, and epithelium, but are not filled. The lining epithelium is swollen. This stage lasts a few hours, but sometimes is prolonged to a few days. (2) *Red hepatization:* The lobe is much enlarged, shows grooves from the ribs; the pleura is coated with fibrin; the lung is firm, friable, does not crepitate, sinks, and is dark red. The cut section is dry, granular, and reddish brown. Microscopically the air-cells, bronchioles, and in massive pneumonia the larger bronchi also, are distended with an exudate of red and white blood-cells, epithelium, and fibrin, and contain many bacteria. The lining epithelium is swollen; the lung-tissue also shows an exudate of cells and fibrin. (3) *Gray hepatization:* The exudate loses its color and softens while the lung is still solid. Microscopically the red cells are not seen. (4) *Resolution:* The products of inflammation disintegrate and are absorbed by the lymphatics. The lung again is filled with air and becomes normal. Consolidation usually appears on the first or second day. Resolution begins at defervescence and is completed within a few days. The rest of the lung is much smaller than normal, and may be congested or edematous. The larger bronchi of the affected lobe are congested and contain frothy liquid. The bronchial nodes are inflamed. Cell degeneration is present in the various organs. Leukocytosis and often pneumococci are present in the blood. The right side of the heart often is distended and full of firm clots at autopsy.

Symptoms.—*Incubation* probably from a few hours to three or four days. The *onset* is very abrupt with a severe chill (p. 23), rapid rise of temperature, and other febrile symptoms; prostration is marked. The temperature continues with slight remissions for about a week and falls by crisis. Marked leukocytosis is present from the onset to crisis. The respiration-pulse ratio is 1:2 or larger. Sharp pain on the affected side of chest with inspiration and coughing is present soon after the onset, but grows less after three or four days. There is a short cough at first, but later a harder cough. The sputum at first is scanty or absent. Soon it becomes very tenacious and rusty. Microscopically it shows red and white blood-cells and pigment-granules, epithelium, granular matter, mucus, and bacteria. Near the crisis it may be mucopurulent and more abundant. Chlorids are often absent from the urine; they usually reappear about a day before the crisis.

Physical Signs.—Inspection: Anxious expression; one cheek often flushed or dusky; herpes on lips and nose is common after second day. Breathing is rapid (30–40 or more); shallow, jerky inspiration, grunting expiration; more restricted on affected side. Alæ nasi dilate. Over the affected part of the lung the following signs are found during *congestion: Palpation:* fremitus slightly increased. On *percussion* the note is higher and shorter, or tympanitic, or less loud. *Auscultation:* breathing and voice usually feeble and indistinct, sometimes harsh, or on deep breathing bronchovesicular. There may be subcrepitant râles. *Stage of consolidation: Palpation:* increased fremitus. *Percussion:* dulness or flatness; increased resistance to finger. *Auscultation:* bronchial voice and breathing. *Râles:* pleural crepitations, or fine moist râles. Moist râles are often larger and more numerous toward the end of this stage. These signs usually appear on the first or second day over a small area and spread to an area somewhat greater than that of a normal lobe. *Resolution:* All signs gradually return to normal. Fremitus lessens, resonance returns, but a slightly impaired note may persist several weeks, breathing and voice become bronchovesicular, then normal. Crepitant, subcrepitant, and larger moist râles are abundant. This stage usually lasts about three or four days. Over the rest of the affected lung during congestion the signs are usually normal. During consolidation fremitus may be slightly increased, resonance may be tympanitic, and breathing and voice harsh. The heart-rate is about 100–120, the first sound at the apex is shorter, the pulmonary second sound is accentuated, the pulse soft, sometimes dicrotic. Splenic dulness is enlarged; the edge of the spleen may be felt. *Irregular physical signs:* Signs of consolidation may appear late or sometimes not at all. Resonance may be tympanitic throughout; fremitus, breath- and voice-sounds may be very faint or absent (massive pneumonia). Signs of consolidation may persist several days or weeks after defervescence and the disappearance of other symptoms (delayed resolution). Râles may be absent. There may be a rough friction-sound which may persist.

Sweating may be marked throughout. Weakness of the heart causes a rapid pulse, lessening of the second pulmonary sound, embryocardia, and edema of lungs. Death from sudden cardiac paralysis is common. Leukocytosis may be absent in the worst cases or in very mild ones. Cough may be absent. Sputum may be very bloody at first. It may be thin, liquid, dark brown (prune juice) in bad cases. It may be absent. *Temperature:* There may be no chill, but a slow rise. A day or two before the crisis there may be a temporary fall of 2 or 3 degrees (pseudocrisis). There may be a precritical rise of 2 or 3 degrees. The crisis occurs from the third to the twelfth day. The temperature sometimes falls by lysis. Hyperpyrexia may be present. *Cerebral symptoms* may resemble meningitis or acute mania or delirium tremens; or there may be apathy at first and the typhoid state after a few days.

Complications. — Parotitis; croupous colitis; jaundice (with green sputum); peritonitis. Pericarditis is common, especially in double and left pneumonias. Usually it is fibrinous, sometimes serous, less often purulent. Malignant or simple endocarditis. Thrombosis and embolism. *Pleura:* Fibrinous pleurisy is usual, but it may be so extensive as to suggest the term pleuro-pneumonia, and to lessen the intensity of fremitus and auscultatory sounds. Pleurisy with effusion develops late, sometimes in convalescence. When it is present lysis is common, leukocyosis lessens, but persists, and the physical signs gradually change to those of liquid. Empyema resembles serous pleurisy, but the fever is more persistent and remittent; there may be sweats and chills; leukocytosis is marked. Pleurisies are common complications. Abscess or gangrene of lung is rare. Meningitis and neuritis are rare. Arthritis, like rheumatism, may appear at any time. The pneumococcus may be found in any of these inflammations.

Convalescence is usually rapid unless interrupted by a complication.

Varieties.—Double pneumonia involves both lungs. In massive pneumonia the bronchi are filled with inflammatory products. Migratory pneumonia involves lobes in succession. In central pneumonia consolidation reaches the surface late or not at all; physical signs may not suggest consolidation, but general symptoms are quite typical. In infants there is a convulsion at the onset; physical signs develop late and are often atypical; the temperature is more remittent and lysis is more common; the cerebral symptoms are more marked; no sputum is seen. In the aged the onset is insidious; temperature, lung symptoms, and physical signs are slight; the chief symptom is prostration. In alcoholics the onset is insidious; temperature and lung symptoms are slight or even absent, but physical signs are usually found if looked for; active delirium is present. Terminal pneumonia in any bedridden exhausted person may cause death, with nothing to suggest pneumonia; physical signs if looked for may make the diagnosis, but they are apt to be atypical. Other secondary pneumonias, in any acute disease, are often due to a mixed infection with pus cocci. Pneumonia of influenza is apt to have a prolonged stage of congestion and imperfect consolidation, with tardy and atypical signs; the temperature is less regular and crisis is less common; other inflammations of the respiratory tract are often present. Larval or abortive pneumonia lasts only a day or two. Asthenic or typhoid pneumonia has general symptoms of intense intoxication and the typhoid state in two or three days; lung symptoms and signs are indistinct or possibly absent.

Progress.—The mortality is 20 to 30 per cent. It is worse in the epidemic form, with age, in persons previously weak and ill, in massive pneumonia or pneumonia of more than one lobe, in complicated cases, alcoholics, cerebral cases, and secondary cases. *Causes of death:* exhaustion, asphyxia, edema of the lungs, or sudden or gradual heart failure.

Treatment.—*Preventive:* Disinfect sputum and things soiled with it. General management as in other fevers (p. 25). *Special treatment:* Antipneumococcic serum may be used. Its effect is not agreed upon. Venesection is indicated early in full-blooded patients with high-tension pulse, and later with cyanosis and weakening second pulmonary sound. Baths, as in typhoid, or with water at 90° F., are used in toxic cases and alcoholics. Salt solution intravenously or by hypodermoclysis is useful in toxic cases or with failing heart. *Locally:* Cold compresses or poultices to the chest over the involved lung relieves pain. Creasote carbonate, ½–1 drachm daily, is said to abort or mitigate. Oxygen by inhalation is used for cyanosis and dyspnea. The standard drugs are strychnin, alcohol, and digitalis. Many others are used. In symptomatic treatment avoid depressing drugs, as the heart is the chief source of danger.

QUESTIONS.

Give the etiology of lobar pneumonia.

Describe the lesions (by stages).

What are the symptoms (including, of course, the physical signs) of a typical, uncomplicated case?

What are the more important complications and the symptoms caused by them?

Give the characteristic features of pneumonia in young children, in the aged, in alcoholics, and of terminal pneumonia.

Discuss the prognosis.

What is delayed resolution? Its symptoms?

Give the details of the routine treatment, and symptomatic treatment.

DIPHTHERIA.

Etiology.—Klebs-Löffler bacillus is found in the exudate and false membrane, in the mouth, throat, and nose of the ill. Sometimes virulent or non-virulent forms are found in the nose and throat of healthy persons, especially those who have been near patients. It is given off in the sputum, discharges from the nose, and in coughing; lives in rooms and fomites for months; it enters the body probably with the breath. Diphtheria is very contagious during the active stage, and perhaps until bacilli have disappeared from the mucous membranes. *Predisposing factors:* Mostly in children; in cold weather; endemic in cities; epidemics are common and may be extensive; sporadic cases occur. It is common with scarlatina; is disposed to by lesions of mucous membranes and by general lack of condition. Immunity for a few weeks follows the injection of 300 to 500 units of antitoxin. An attack gives immunity for a few weeks only, if at all, and may then be followed by greater susceptibility. Second attacks occur.

Lesions.—A croupous inflammation (p. 18), with diphtheria bacilli alone, or combined with ordinary pus cocci and other bacteria. The membrane may be infected with putrefactive bacteria and become dark and fetid. The process usually starts in the tonsil or nasopharynx, sometimes in the larynx. It then spreads more or less over these structures, the palate, or down the trachea. In surrounding tissues are a variable amount of edema and exudative inflammation. Lymph-nodes are inflamed. Cell-degenerations elsewhere attack, especially the kidneys, heart, and nerves.

Symptoms.—*Incubation,* usually about two days, but may be ten days. Prodroma are not important. The *onset* is not striking. The *general symptoms* are fever of moderate grade, an irregular temperature (101°–102° F.); the heart is relatively rapid and often weak; febrile albuminuria is very common and early; apathy and prostration are marked; the patient looks pale and sick. The fever lasts a week or ten days, and goes by lysis. Leukocytosis is present, increasing for three or four days, and then disappearing with the other symptoms. *Local symptoms:* At first redness; after a day a pale gray membrane appears, spreads, and becomes thicker and opaque. It is stripped off with difficulty and leaves a bleeding surface. If removed, the membrane reforms quickly. The membrane may putrefy, becoming dark brown or green or nearly black. In recovery the membrane grows thin, curls at the edges, is cast off in imperceptible masses or in flakes of various sizes. It leaves a red surface that fades in three or four days. Corresponding lymph-nodes are large, tender, and painful; and seldom resolve with the main lesion. *Faucial cases:* These may look like follicular tonsillitis at first, but the membrane spreads beyond the tonsil, to palate, uvula, and pharynx. The tonsils may be much swollen, the uvula edematous; there are sore throat, dysphagia, and a thick voice. These cases may be mild. After a week healing begins, and in three or four days more the patient is convalescent. *Pharyngeal cases* are similar, but the membrane appears on the back of the pharynx and tends to spread to the nose. *Laryngeal cases:* A laryngoscope is necessary to see the membrane at first, but later it may spread to the pharynx or tonsils, also down the trachea. Cultures from the pharynx often show the bacilli on the first or second day. There are increasing dyspnea, with signs of insufficient aëration of the blood, cyanosis, weakening heart, even marked asphyxia, varying with the size of the larynx, the amount of membrane, and the surrounding swelling. Pieces of membrane may be coughed up, giving temporary relief, but the membrane re-forms in an hour or two. Laryngeal cases are serious. *Nasal cases* have snuffles, sneezing, mouth-breathing, a thin discharge from the nose, which becomes brown or red and excoriates the alæ nasi and lip. The membrane, often putrid, is seen in the nose. It may extend to accessory sinuses or to the conjunctiva and pharynx. Lymph-nodes are much swollen. General toxemia is marked. These cases, more common in children, are serious. (Occasionally there are nasal cases with marked local symptoms and membrane, but little or no general disturbance, running a subacute course of several weeks.) *Unusual symptoms:* Diffuse erythema, urticaria, or purpura (even when antitoxin is not used). Much vomiting, diarrhea. Secondary anemia sometimes is severe and persists for weeks.

Complications.—Parotitis. Bronchopneumonia, especially in laryngeal cases. Atelectasis. Neuritis appears late or in convalescence. In the cardiac nerves it may cause sudden death, or a progressive heart weakness that may be fatal after a few hours, or a weak, slow pulse. Neuritic paralysis may attack the muscles of the soft palate, pharynx, larynx, eye, limbs, or trunk. Recovery usually follows, but paralysis of the respiratory muscles may be fatal. Otitis media is common. Nephritis, late or in convalescence, is common.

Varieties.—Besides those already mentioned there are *latent* *cases* with no membrane, but a catarrhal inflammation of the throat; Klebs-Löffler bacilli are present; general symptoms are mild and last a day or two. *Unusual sites:* Conjunctiva, vulva, prepuce, wounds or raw places on the skin. *Malignant cases:* Often in nasal cases there are general symptoms of intense toxemia, typhoid state, very weak heart, skin rashes, sometimes bleedings, and death in a week. Sometimes there is cellulitis of the neck and head.

Prognosis.—The mortality varies much in different epidemics. It used to be 20 to 40 per cent.; with antitoxin it is one-third to one-half of this. The bad cases are laryngeal cases (in proportion to the smallness of the larynx), nasal cases, cases with extensive membranes, putrefaction of the membrane and necrosis of tissues, much vomiting, rapid, weak heart, pneumonia, severe toxic symptoms, much albuminuria, and malignant cases. The chief causes of death are laryngeal stenosis, septic intoxication or infection, heart failure, and pneumonia.

Diagnosis.—Cultures are the most important.

Treatment.—*Preventive:* Rigid isolation of the patient and those with Klebs-Löffler bacilli in the throat until two cultures show absence of bacilli. Disinfection of contaminated discharges and of everything in contact with the patient, and of the room. Immunizing injections of antitoxin to those exposed. Special care of catarrhs in children. Cultures should be taken from suspicious throats. *General management* as in fevers. Because of the heart, the child should stay in bed for two weeks after defervescence. He should not nurse. It may be necessary to feed with a catheter or stomach-tube. Stimulants are often needed—alcohol strychnin, and morphin. *Local treatment:* The throat is sprayed or swabbed with antiseptic solutions, such as Löffler's solution (menthol, 10; toluol, 36; liq. ferri chloridi, 4; alcohol, 60). Calomel fumigation is used. Ice on the neck is used for cellulitis and in laryngeal cases. For laryngeal obstruction intubation is used and the child is fed by gavage or with the head low. The tube is left in from a few days to two weeks. Tracheotomy may be needed. *Antitoxin:* A horse is made immune by repeated, increasing injections of the virulent cultures and toxins. His blood-serum is then collected (Behring's unit is the following: A mixture of 0.1 of this unit of antitoxin with 10 times the minimum fatal dose of toxin for a 250-gram guinea-pig, is harmless when injected into such a guinea-pig). As soon as the diagnosis is made by culture, or earlier in severe cases, inject into the buttocks, thigh, flank, or subscapular space 1000 to 4000 units. Repeat the dose in twelve to twenty-four hours, and later if necessary. The maximum dosage is indefinite, but 100,000 units seem to have saved some cases. In very bad cases intravenous injection has worked well. *Results:* As a rule, the toxic symptoms lessen promptly, the local swelling and the membrane vanish, and the child lives. The effects are less favorable after the third day. Rashes due to horse serum, not to antitoxin, and joint-pains follow in some cases after a day or two. They are usually erythema or urticaria.

PSEUDODIPHTHERIA.

Etiology.—This condition is not caused by the pseudodiphtheria bacillus, but by the streptococcus. It is sometimes primary, but more often secondary to some infection, such as scarlatina, measles, whooping-cough, or typhoid. The lesion is a croupous inflammation of the fauces, pharynx, and larynx.

Symptoms.—The temperature, in primary cases at least, is often higher and the toxemia less than in diphtheria. The local symptoms are very like those of diphtheria, but the membrane may be less adherent, is more often limited to one tonsil, and is much less apt to attack the larynx.

Primary cases without complications have a good **prognosis,** but laryngeal cases may be dangerous. Secondary cases often mean a severe or fatal illness.

The **diagnosis** is best made by culture.

Treatment.—Isolation is a disputed point. Antitoxin is not useful. Otherwise the treatment is like that of diphtheria.

QUESTIONS.

Discuss the etiology of diphtheria.

Give in detail the lesions.

What are the general symptoms of diphtheria?

What are the local symptoms of throat cases, of nasal cases, of laryngeal cases?

Where else may diphtheria occur?

What are the complications?

Discuss the prognosis, and mention the serious symptoms and the causes of death.

Give the details of disinfection during and after the disease, and the degree of isolation to be practised.

Discuss antitoxin, its preparation and its immunizing and curative uses.

Give the other treatment.

Discuss the use of cultures in nose and throat cases and in laryngeal cases.

Pseudodiphtheria: give its etiology, symptoms, prognosis, and treatment. Differentiate it from diphtheria.

SEPTIC DISEASES.

Sapremia.—This is caused by the bacteria of putrefaction growing in dead matter, such as placental tissue in the uterus or a blood-clot, or in matter in a wound or cavity, or in injured tissues. They form poisons which are absorbed and cause the symptoms. *Lesions:* Rigor mortis is early, less marked and less lasting; putrefaction sets in early; the blood is dark, poorly clotted, stains the endothelium red, and there are extravasations in many places; the spleen is large, soft, and dark; there are degenerations of various cells. There is also the primary focus. *Symptoms:* The incubation is very short, perhaps a few hours. The general symptoms are those of fever, which may be very slight and of short duration, or of any severer grade up to the typhoid state with death after a few days. Leukocytosis is regularly present, and is proportional to the poisoning; it may be absent in very mild cases or very intense cases. Anemia is usually rapid and proportional to the severity of the case. *Course:* If the focus is cleaned and kept clean, the poison is rapidly excreted and the symptoms disappear in a few hours or a day or two. Convalescence is rapid and complete. The *prognosis* depends upon the resisting power of the patient (being therefore worse in the weak and old) and upon the removal of the cause. Usually it is good even in severe cases. *Treatment: Local:* Remove the cause surgically. *General:* The treatment for fever.

Septicemia.—Bacteria enter the body and thrive in the living tissue, forming toxins that cause the symptoms. There may be a focus of infection with local inflammation or necrosis, but most of the poison is not formed there, but by the bacteria that are in the blood. If this local lesion is not discoverable, the condition is called "cryptogenic septicemia." The bacteria concerned are usually the streptococcus and staphylococci of suppuration, sometimes the pneumococcus, bacillus proteus, bacillus pyocyaneus, meningococcus, gonococcus, etc. The *lesions* are like those of sapremia, with the addition of bacteria in the blood and the local focus. *Symptoms:* The incubation period is a day or two. The onset is often abrupt, with a chill and fever; the temperature may range from 103° to 105° F., with remissions of a degree or two; after a few days bacteria may be demonstrated in the blood by cultures; leukocytosis is present; there may be a petechial rash or other rashes, or slight jaundice. The *course* may be mild or severe. Death in the typhoid state may follow after a week or two. The *prognosis* is worse than that of sapremia because the cause is disseminated in the blood.

Pyemia.—The bacteria get into the veins, thrombi form around them, and embolism results, with abscesses at the points of lodgement. The *lesions* are those of septicemia with the addition of metastatic abscesses, usually in the lungs and spleen, but often elsewhere. *Symptoms:* The incubation period is about a week, the onset is abrupt; there is a series of paroxysms of chill, fever (103°–105° F.), and sweating. These occur more or less regularly once or twice a day or less frequently. After sweating the temperature may be 100° F. or normal or subnormal. Skin rashes are common. There are leukocytosis and the symptoms of the metastatic abscesses. If infection is through the portal vein, it causes pylephlebitis and the abscesses are in the liver. If septic thrombi form on the heart valves, arterial pyemia follows and abscesses are very general. Pyemia is usually fatal in one to three weeks. The *treatment* of septicemia and pyemia are, like that of sapremia, surgical.

QUESTION.

What are the differences in causation, lesions, and symptoms of these three conditions?

ACUTE ARTICULAR RHEUMATISM.

Etiology.—The specific cause is not known, but bacteria may frequently be found in the joints. The point of entrance is not known, perhaps it is the tonsil. *Predisposing factors:* It is not rare in children after two years, and is most common up to forty. It is more common in young girls and adult males, among those working in damp places, with changeable temperature, in temperate climates, in cold weather, after exposure, with a hereditary tendency. Epidemics occur. It is endemic in some buildings. An attack increases susceptibility and repeated attacks are common.

Lesions.—There is an exudative inflammation of periarticular structures with synovitis, and with more or less albuminous, turbid liquid in the synovial sacs. Resolution usually is complete. Suppuration almost never occurs. There are various degenerations, especially in the voluntary and heart muscles. The lesions of the heart and pericardium are described elsewhere.

Symptoms.—The *incubation* period is unknown; there may be prodroma, such as malaise or discomfort in the joints, or tonsillitis for a day or two. The *onset* is usually abrupt, sometimes with a chill or chilliness. There is fever of an irregular course (101°–104° F.), with marked remissions or intermissions. There is much sweating, often acid, with sudamina and miliaria. The mind is usually clear. Anemia develops rapidly and there is marked leukocytosis. The *joint-symptoms* may be the first symptoms or may appear after a day or two. The joint is swollen, very tender, very painful on motion; the skin is hot and often red. These symptoms develop in a few hours; last, in one joint, one to several days; and may disappear in a few hours. Several joints are inflamed together or successively. In a rheumatic attack the same joint may be attacked more than once. The joints involved are usually the knees, ankles, elbows, and wrists, less often other joints or the synovial sheaths. In children the spine also may be involved for several weeks or months. All the joints may clear up for a day or two, and then a relapse may occur. Especially in children subcutaneous nodules may appear on tendons and ligaments of the hands, wrists, or the spine and persist several weeks or months.

Complications.—Erythemas, especially erythema nodosum, or purpura. Tonsillitis. Endocarditis, especially mitral, is very common, particularly in children. Embolism, myocarditis, and sudden death are rare. Pericarditis, usually serous, is very common. Pneumonia and pleurisies may occur. Hyperpyrexia is not common. In it the temperature rises rapidly to 106° or 109° F., with marked delirium and prostration; if not treated, it is almost always fatal in three or four days; it is often preceded by a lessening of the joint-symptoms. Chorea seems to be closely related to rheumatism and is common in children.

Course.—The attack drags on with remissions or intermissions for four to eight weeks, or even longer.

In *children* tonsillitis is common, the fever and joint-symptoms often are not marked. Endocarditis and pericarditis are very common.

Sequels.—Chronic endocarditis. Stiffness or subacute symptoms in one or more joints which may last several weeks. Anemia. Chorea in children.

Prognosis.—A few cases are fatal from hyperpyrexia, heart failure, or pneumonia, otherwise the prognosis of an attack is good. There is the danger of other attacks and of the sequels.

Treatment.—*General treatment* as in other fevers. *Drugs:* Salicylic acid compounds in large doses until the pain and joint-symptoms cease; then in smaller doses for a week. Some cases do better with alkalies in large enough doses to make and keep the urine alkaline. *Local treatment:* Methyl salicylate, salicylic acid, or sodium salicylate; heat or cold. Cotton and a loose bandage. For hyperpyrexia persistent cold bathing. *Convalescence:* The patient should be kept in bed for a week after defervescence. Solid food should be resumed slowly. For the anemia, iron. For the stiffness of the joints, massage and warm baths.

QUESTIONS.

Give the etiology of acute rheumatism, the details of the joint-lesions, the symptoms of the ordinary uncomplicated case.

What are the common complications and the sequels? Discuss the prognosis and give the treatment.

GONORRHEAL ARTHRITIS.

Etiology.—It follows gonorrhea of the genito-urinary organs, or of the eye, as an early or a late complication. It is more common in males. It is due to the gonococci in the joints, or perhaps to poisons absorbed from the primary site.

The **lesion** is an exudative inflammation of the synovial membrane and surrounding structures with serofibrinous or turbid effusion often containing gonococci. Purulent effusion is rare. The effusion persists a long time and fibrous ankylosis often follows. It involves the joints, synovial sheaths, and bursæ. It often is monarticular in the knee, wrist, or ankle, or may involve many joints in the hands, feet, or spine.

The **symptoms** are usually late in the gonorrhea. *Acute cases:* The local symptoms are acute and intense redness, swelling, pain and tenderness in one or more joints. If several joints are attacked all but one or two usually recover in a few days. The fever and general symptoms are relatively slight. *Chronic cases:* There may be hydrarthrosis, usually monarticular, often but slightly painful, or inflammation of the synovial sheaths, the bursæ, and the periosteum, with more severe pain. Both forms leave restricted movement.

Complications.—Rarely pus forms in the joints. There may be other signs of systemic gonorrhea, such as simple or malignant endocarditis, fibrinous or serous pericarditis, serous pleurisy, or general sepsis or pyemia.

Prognosis.—It is apt to last for months and leave a stiff joint.

Treatment.—*Local:* Incision and irrigation of the joint; or immobilization, compression, counterirritation, or dry heat. In chronic forms use massage and passive motion. General tonic treatment and the treatment of the primary gonorrhea are needed.

QUESTIONS.

Discuss the etiology of gonorrheal arthritis.
What are the lesions?
What are the symptoms of acute and chronic cases?
Discuss the prognosis and treatment.

ASIATIC CHOLERA.

Etiology.—Spirillum choleræ asiaticæ is found in intestinal contents and superficially in the mucous membrane of cholera patients; sometimes in the stools of those merely exposed to cholera. It thrives in contaminated water; lives on many foodstuffs and in the soil; is killed by drying. Infection occurs by means of water or food or by flies. The disease is endemic in places, spreads with travel in epidemics; attacks all ages and both sexes; especially those in unsanitary surroundings and with digestive disturbances. Cold weather checks an epidemic. Immunity seems to follow injections of cultures or toxins.

Lesions.—Rigor mortis is early, marked and persistent; the tissues are shrivelled; putrefaction is delayed. The intestines are filled with "rice water" containing many comma bacilli, flakes of fibrin, and blood-clots; the mucous membrane shows acute catarrh with ecchymoses; the muscular coat is relaxed; the serous coat congested; the lymphoid tissue swollen. A croupous inflammation, chiefly in the colon, may be found. The stomach shows less marked changes. The liver and kidneys are congested and their epithelium is degenerated. There is degeneration of the heart and other muscles.

Symptoms.—*Incubation period:* Usually three to five days. The stages of the disease are: premonitory diarrhea, serous diarrhea, collapse, reaction. (1) *Premonitory diarrhea* shows the symptoms of a severe diarrhea with colic, sometimes cramps in the legs, slight fever, and prostration. It may last from a few hours to three days, or may be absent. (2) *Serous diarrhea:* In this stage the skin is cold, wrinkled, and livid; the tongue coated; there are thirst, anorexia, vomiting of rice-water material; several large rice-water stools a day, passed without pain. The heart is fast and weak; the pulse hardly palpable. The blood is thicker, less alkaline, and may show an excess of red cells and leukocytes. The rectal temperature is 103° F. or more; the skin temperature 95° or 96° F. The mind clear or dull. The urine is diminished or suppressed; it contains albumin and casts. Cramps and twitching in the limbs, especially in the calves, are marked; prostration is extreme; the body looks shrivelled. This stage may last half a day. (3) *Collapse:* The vomiting and diarrhea lessen or stop. There is marked collapse or perhaps coma. This stage lasts a day or less. (4) *Reaction:* All the symptoms improve rather promptly. After this there may be (*a*) a relapse into the previous stage, or (*b*) cholera typhoid—*i. e.*, the typhoid state often with erythema or petechiæ, and secondary infections; (*c*) acute uremia.

Complications.—Secondary infections; croupous inflammations of any mucous membrane, especially the throat, colon, and external genitals; parotitis; bronchitis; pneumonia; pleurisy; or general septicemia.

Course and Varieties.—Convalescence may follow the premonitory diarrhea. "Cholerine" is like a mild second stage, which lasts about a week and is followed by recovery. In "cholera sicca" the patient collapses, has all the symptoms of cholera except the stools, and dies after three or four hours. The gut is paralyzed and filled with rice water. Fulminating type: The patients become comatose and die within a few hours. Death in the *regular cases* may occur in the stage of collapse, or in the stage of reaction. Recovery may follow the first or second stage, rarely the third stage. The mortality is from 20 to 80 per cent.

Treatment.—*Preventive:* Isolation; rigid disinfection of all stools, vomitus, and contaminated objects, which must be continued for a week of convalescence or until cultures show no comma bacilli in the stools. Those exposed should use only boiled water and cooked food, and should try to keep a perfect digestion. Immunizing injections are used. Slight disturbances of health should be treated at once. *Treatment of the disease:* Enteroclysis with warm tannic acid solution or salt solution, four times a day or oftener, is recommended.

The diet is liquid. Morphin, alcohol, ammonia, and strychnin are used. When the drain of liquid is marked, salt solution is injected intravenously or subcutaneously, and external heat is needed.

QUESTIONS.

Give the etiology, the lesions, the stages, and their symptoms of cholera.

Discuss the prognosis and give the treatment.

DYSENTERY.

The etiology and classification of dysentery have not been thoroughly worked out.

(1) **Acute Catarrhal Dysentery.**—The specific cause is not known. It occurs at all ages, but especially in children, and in unsanitary surroundings. *Lesions:* The colon and sometimes the ileum show an acute catarrh with excessive mucous secretion; the lymphoid tissue is inflamed and often necroses with the formation of ulcers; sometimes rather large irregular ulcers may form. *Symptoms:* It usually begins with an ordinary diarrhea and a little fever. Within two days *dysenteric symptoms* appear. These are intestinal colic, rectal tenesmus, and very frequent small stools containing mucus, a little blood, pus-cells, and swollen epithelium, but no fecal matter and without fecal smell. They contain various bacteria. The abdomen is flat and hard, and it is tender along the colon. There are great thirst, anorexia, sometimes vomiting; the temperature may be 102°–104° F.; prostration is marked. After a week improvement begins; the stools become less frequent and free from blood; then more opaque, and finally fecal matter appears and the stools become normal. The other symptoms subside gradually. These cases usually recover.

(2) **Croupous or Diphtheritic Dysentery.**—The specific cause is unknown. The cases are primary or secondary. (*a*) *Primary cases:* These are sporadic. *Lesions:* There is a croupous inflammation of the colon, with more or less thickening of the wall of the gut. The necrotic membrane varies from streaks at the edges of the folds of the mucous mem-

brane to a very extensive necrosis involving the whole thickness of the mucous membrane. Mild cases involve chiefly the flexures and rectum; the severest cases involve the whole colon. Mild cases heal by granulation, but severer cases show no attempts at healing. *Symptoms:* The onset is abrupt, with the general symptoms of marked fever, 104°–105° F. In three or four days the typhoid state develops. The abdominal symptoms are colic, constant pain, tenderness, tympanites; the abdomen feels hard; the stools are fewer than in the preceding form, but are larger and may contain some fecal matter and sloughs, beside the blood, mucus, and pus. Death usually follows in from one to three weeks. Sometimes a slow recovery takes place. (*b*) *Secondary cases:* The bacteriology is unknown; it is a terminal infection in chronic heart or kidney disease, in cachexias, and in acute infectious diseases, such as pneumonia, typhoid, or tuberculosis. The *lesion* is like that of the primary case with less diffuse thickening of the gut. The symptoms may not suggest the colon, but usually there are three or four large loose stools a day containing some blood and mucus. Sometimes there are the typical stools and symptoms of dysentery.

(3) **Specific Dysentery.**—The specific cause is probably Bacillus dysenteriæ of Shiga, or some of its varieties. It resembles the typhoid bacillus; is found in the stools and intestinal walls of the patients, and in drinking-water. It is agglutinated by dilute solutions of the blood-serum of patients. An antitoxic serum has been used with promising results. The disease is epidemic in armies and various countries. It is endemic in many places, especially in the tropics and Japan. Recent research seems to show that this is the usual dysentery occurring in epidemics throughout the temperate zone in late summer and fall. Probably it is transmitted by water, and is predisposed to by unsanitary surroundings and digestive disturbances. *Lesions:* In the colon and sometimes in the ileum the mucous membrane is swollen and firmer. There are more or less ecchymosis and superficial necrosis of the mucous membrane. The lymphoid tissue is swollen. In the most intense cases there is extensive necrosis. *Symptoms:* *Acute cases:* The onset is abrupt, with colic, diarrhea with mucus in the stools, and moderate

fever. After a day the typical abdominal symptoms and stools of dysentery develop; the fever is high, 103° to 104° F.; the typhoid state comes on and the patient may die in three or four days or later. The favorable cases last two or three weeks and recovery is slow. The *subacute cases* are less intense, but emaciation becomes marked; they last a few months, and end in death or recovery. Relapses are common. Almost all cases show the agglutinative reaction: and all have the bacteria in the stools. The mortality of the acute cases is 10–30 per cent.

(4) **Amebic Dysentery.**—Amœba dysenteriæ is present in the stools and the lesions, and is probably the cause. The ameba is a single-celled animal, 15 to 20 μ in diameter, with a clear outer zone and granular central portion. The central portion contains the nucleus and vacuoles. The ameba is found in the stools and shows active movements; it often contains ingested red blood-cells. Many cases in armies and epidemics are of this group, chiefly in hot climates, and perhaps are transmitted by water. Inexplicable sporadic cases occur in more northern climates. *Lesions:* The chief lesion is masses of proliferated connective-tissue cells in the submucous coat, forming nodules that project from the surface. These necrose at the center, and form ulcers with undermined edges; the ulcers vary in size and number; they may run together and intervening mucous membrane may slough. There is also catarrh of the mucous membrane. The lesion may involve the ileum. Chronic cases may show scars of healed ulcers. *Symptoms: Acute cases* have the usual dysenteric symptoms with amebæ in the stools. Death may follow in a week; or recovery after two or three weeks; or the ulcers after sloughing may lead to emaciation and diarrhea, with death in a few months. *Chronic cases* may follow the acute or subacute cases, or be chronic throughout. There are attacks of diarrhea or dysentery, with the amebæ in the stools. Errors in diet are apt to bring on an attack. The disease lasts for years.

Complications of Dysentery.—At times there may be a large intestinal hemorrhage, peritonitis with or without perforation; anemia; endocarditis; pericarditis; phlebitis; pneumonia;

paraplegia due to neuritis; nephritis; or arthritis. In amebic cases there is often abscess of the liver or lungs.

Prognosis.—The catarrhal cases usually recover. Diphtheritic cases seldom recover. In specific dysentery 10 to 30 per cent. die. Amebic dysentery causes a few deaths in the acute stage, but tends to become chronic. Dysentery is apt to leave a patient in poor health for years. The cause of death usually is asthenia, less often, some complication.

Treatment.—Rest in bed and a milk diet. A mild purge may be given at the onset. *Ipecac treatment:* After a few hours of fasting, morphin gr. ¼ to ⅓ is given, and half an hour later ipecac gr. xx–lx in one dose. *Saline treatment:* Sodium sulphate, ʒj, four to eight times a day, for two or three days, or until the stools contain no mucus or blood. *Irrigation of the colon:* The solutions used are from 2 to 8 pints of silver nitrate, 0.1–0.2 per cent.; tannic acid, 1 to 2 per cent.; and especially in amebic dysentery, quinin, 0.1–0.02 per cent. This is repeated two to five times a day. Antidysenteric serums give promising results when the Shiga bacillus is present.

QUESTIONS.

What are the symptoms of acute catarrhal dysentery?

Give the etiology, lesions, symptoms, and prognosis and treatment of specific dysentery.

Give the etiology, lesions, symptoms, and the chief complication of amebic dysentery.

Describe Amœba dysenteriæ.

What special diagnostic points are there in specific dysentery and amebic dysentery?

Give the general treatment of dysentery.

Describe the treatment with ipecac, the saline treatment, the irrigation treatment.

YELLOW FEVER.

Etiology.—The specific cause is unknown; it probably occurs in the blood. If a certain mosquito (*Stegomyia fasciata*) bites a yellow fever patient and after about two weeks bites a susceptible person, that person will develop yellow fever in from two to six days. This mosquito breeds in small bodies of water, and bites in the daytime. Blood from a patient injected into a susceptible person gives the disease. The disease is endemic in places in the West Indies and along the Gulf of Mexico. It spreads in epidemics in warm weather to places further north and south; hard frost checks the epidemic. There is greater susceptibility in children, in males, in white races, and among new arrivals in epidemic places. An attack gives permanent immunity.

Lesions.—There is general congestion with a tendency to bleeding and degeneration, most marked in the liver and kidneys. The blood is thick and dark, and is laked.

Symptoms.—*Incubation period*, including the mosquito part of the cycle is two or three weeks, but after the bite of the mosquito from two to six days. The stages are: (1) the invasion or initial stage, (2) remission, (3) the hemorrhagic stage. (1) *Stage of invasion:* The onset is abrupt, with chill, fever (102° to 104° F.), headache, and severe pains in the loins and legs. There are three cardinal points: (*a*) intense congestion and slight jaundice of the face, especially in the conjunctivæ; (*b*) with a stationary or rising temperature, the pulse-rate falls; (*c*) early albuminuria. The first stage lasts from one to three days. (2) In the *remission* all the symptoms abate, the temperature falls several degrees. Convalescence may follow, or after a day or less the third stage begins. (3) The fever returns with a high temperature and rapid, feeble pulse; the typhoid state or collapse comes on, but delirium is rare. There are marked jaundice, hemorrhages into the skin, mucous membranes, and elsewhere, with black vomit and tarry stools, partial or total suppression of urine, and often acute uremia. This stage lasts a few days, and death in collapse is very common. Recovery by lysis follows rarely. The total duration of the disease is about a week. Convalescence is apt to be slow, and may be *complicated* by parotitis, various abscesses, or diarrhea.

Varieties.—*Mild cases:* Merely a slight illness for one to three days; these cases are not diagnosed except in times of epidemic. *Malignant cases* die in the first stage, after two or three days. The general mortality is from 10 to 50 per cent.

Treatment.—*Preventive:* If a case appears, screen all doors and windows of the house and neighboring houses, and every day kill all mosquitoes that may be in the houses. Screen or destroy all water receptacles in the neighborhood. Attack the mosquito in every way. The official quarantine lasts five days. These measures are remarkably successful. For the *patient* use the general treatment for fever. Nutritive enemata are often necessary. Cold baths are used for the fever, and general symptomatic treatment is indicated.

QUESTIONS.

Discuss the mosquito and yellow fever.
Give the symptoms, the prognosis, and the preventive treatment.

PLAGUE.

Etiology.—Bacillus pestis; it occurs in buboes, the blood, the feces, and the urine; it has been found in the soil and the dust of rooms; it attacks lower animals, especially rats. The mode of infection is probably usually by inoculation, less often inhalation or swallowing. Direct contagion is rare. An epidemic in rats usually precedes one in man. Fleas, flies, or fomites transmit the disease. Dirt and crowding predispose. Plague is endemic in parts of Asia, spreads with travel in epidemics that may be very extensive. Immunity is conferred by the disease, by the injection of sterilized old broth cultures (Haffkine's prophylactic), or of antitoxin from the horse and other animals.

Lesions.—The lymph-nodes are inflamed and may suppurate or become gangrenous; they contain the bacillus in great numbers. There may be bronchopneumonia or a general septicemia, both due to B. pestis.

Symptoms.—*Incubation period,* three to seven days. *Bubonic cases:* Prodroma may be present, such as headache, dulness, or a drunken gait for a day. The *onset* is abrupt with chill, fever (102°–106° F.), marked prostration, and a leukocytosis. In severe cases there may be bleeding into the skin in petechiæ, or blotches (black death), and sometimes in the stomach, intestine, or elsewhere. The *buboes* appear from the second to the fifth day, and are tender painful, and often large; they may suppurate and break through the skin (a favorable symptom) or become gangrenous (a bad symptom), or soften and resolve. The femoral and inguinal nodes are usually attacked. The fever remits in three to five days, but soon rises again, and continues for a week or longer; finally it goes by lysis.

Varieties.—*Pestis minor:* There is some swelling of the lymph-nodes with malaise lasting a week or two. *Pestis major:* (1) The *bubonic type* is the most common; the mortality is about 50 per cent.; death is common from the second to the fifth day; it is rare after the first week. With recovery the convalescence is slow. (2) The *pneumonic type:* There is a bronchopneumonia with bacillus pestis and blood in the sputum. The patients die in two or three days. The mortality is 95 per cent. (3) *Septicemic type:* There is a general infection with the bacilli, but no buboes. Death follows in one to three days. The mortality is about 80 per cent. Some collapse and die in a few hours (*pestis siderans*).

Treatment.—*Preventive:* Rigid isolation is practised for a month after recovery. All discharges and fomites are disinfected. Corpses are destroyed. All rats should be destroyed. Exposed persons should be immunized. These measures and quarantine for a week are very efficient in sanitary communities. *Curative:* An antitoxin obtained from the horse or other animal gives favorable results. Suppurating buboes may be incised. There is the usual general management for fevers and symptomatic treatment.

QUESTIONS.

What are the etiology, the incubation period, the symptoms of bubonic plague?

Give the varieties, prognosis, and treatment.

RELAPSING FEVER.

Etiology.—The specific cause is Spirochæta (spirillum) Obermeieri. This is actively motile, is found in the blood during the fever only, and is not known outside of the body. Predisposing factors are dirt, bad air, and famine. It is moderately contagious and may be transmitted by fomites. No immunity follows an attack. It occurs in epidemics.

No specific **lesions** are known.

Symptoms.—The *incubation* is a week or less; the *onset* is very abrupt, with a chill and fever. On the second day the temperature reaches 106°-109° F. There may be herpes, petechiæ, and jaundice. The blood shows the spirochete and leukocytosis. The fever continues high for about a week; falls by crisis, sometimes with collapse; and after a day or two the patient feels pretty well. On the fourteenth day from the onset he has another paroxysm, a little milder and a day or two shorter. One or two more relapses may follow after the same interval, and then convalescence begins and is slow. The mortality is about 4 per cent.

No special **treatment** is known.

QUESTION.

Give the etiology, the temperature-curve, and the prognosis of relapsing fever.

MALARIA.

Etiology.—The specific cause is a single-celled animal, Hemameba or Plasmodium. Its definitive host is the female of a mosquito (*Anopheles maculipennis*), in which it undergoes sexual reproduction. Its temporary host is man, where it reproduces asexually. The *æstivo-autumnal parasite:* In man the youngest form seen is a clear, colorless, tiny, motile, annular body in a red blood-cell. Later, one part of the ring becomes thicker, like a signet ring. The ring grows to a diameter about half that of a red cell. These are in the peripheral blood. The later stages usually are found in the capillaries of the viscera, especially the spleen and the bone-marrow. The "signet" increases; the rest of the ring disappears, leaving a slightly motile, discoid body, containing a few masses of pigment. The red cell looks shrunken and brassy. The pigment gathers at the center of the organism, and radial planes appear, forming a rosette about two-thirds the diameter of a red cell in size. Along these planes the organism divides into ten to twelve spores, which, with the pigment, become free in the blood. The spores attack other red cells and begin another cycle. This is the asexual cycle, and its duration is variously given as twenty-four to forty-eight or even seventy-two hours. After about a week crescents appear in the peripheral blood, but are more numerous in the viscera. The crescent is longer than the diameter of a red cell (which may show as a pale arc in the concavity of the crescent), is not motile, contains pigment at its middle, and has rounded horns. It is a sexual form, and develops no farther in man. The mosquito ingests them with the blood.

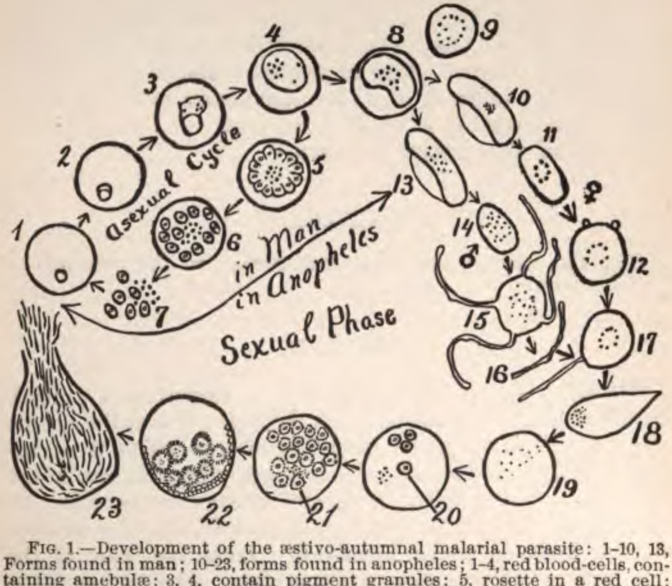

FIG. 1.—Development of the æstivo-autumnal malarial parasite: 1–10, 13, Forms found in man; 10–23, forms found in anopheles; 1–4, red blood-cells, containing amebulæ; 3, 4, contain pigment granules; 5, rosette in a red cell; 6, spores and pigment in a red cell; 7, spores and pigment free in the blood; 8, crescent formation; 9, extracellular sterile form; 10, 13, crescents; 11, 12, macrogametocytes, female gametes; 14, microgametocyte, male gametes; 15, flagellated microgametocyte; 16, free flagellum or microgamete; 17, flagellum fertilizing macrogametocyte, after fertilization called macrogamete; 18, traveling vermicule; 19, young zygote; 20, 21, zygotomeres within zygotes; 22, blastophore; 23, mature zygote containing sporozoites (modified from Rees).

In the mosquito's stomach the crescents develop, and are seen to be of two kinds. They become spherical. One kind, the male, pushes out long, slender flagella, which become separated and move about actively. When one encounters a female body, it penetrates it and fertilizes it. This impregnated body then becomes actively motile and makes its way through the stomach-wall of the mosquito to the outer surface of the stomach. It lodges here and grows larger, a capsule becomes apparent, and within the capsule it divides into many little spheres. Each sphere develops radial striations, and finally breaks up into fine spindles, which fill the capsule. The capsule breaks and discharges the spindles into the body cavity of the mosquito. They then get into the salivary gland, are inoculated into men when the mosquito bites, and resume asexual development. One mosquito may infect more than one person. The development in the mosquito needs from eight to twenty days. In man, after the infecting bite, the incubation period is from five days to two or three weeks in the various forms, being shorter in the æstivo-autumnal and longer in the other forms. The *tertian parasite* in its early stage is a small, clear, highly refractive ameboid ring, in a red cell, and one-third to one-fourth its diameter. It grows, and after about eight hours fine pigment-granules appear in it. They are numerous near the periphery and vibrate actively. The red cell grows larger and much paler, and the parasite grows so as to quite fill it. Finally the pigment gathers near the center, a rosette is formed, and splits up into about twenty spherical spores. These and the pigment become free in the blood. The spores attack new red cells and the cycle is repeated. This cycle takes forty-eight hours, and all stages are found in the peripheral blood. The *quartan parasite* is much like the tertian, but is more refractive, less active, the pigment is abundant and in coarser grains, rosettes are more common in the peripheral blood, the spores are from six to twelve, and at all stages except the earliest it is smaller than the tertian. The cycle lasts seventy-two hours, the red cell is shrunken and darker. The tertian and quartan parasites have large sexual forms. Their development in the mosquito is similar to that of the æstivo-autumnal. The onset of a paroxysm occurs when the spores are set free and attack the red cells.

The essential factors in infection are the mosquito and existing cases of malaria which infect the mosquito. The mosquito bites at night, and is more abundant near the ground than in second stories; its eggs hatch in pools and sluggish streams. Malaria has a much narrower distribution than formerly. The severer forms are endemic in and near the tropics; most cases occur in the spring and fall. The milder forms occur in many places in the temperate zone, mostly in the fall. Natives in malarial districts often harbor the parasite without having symptoms, but they infect mosquitoes. An attack gives no immunity.

Lesions.—*Acute cases* have a large soft spleen and the blood-changes. *Chronic cases* have a large hard spleen, gray from pigment; the liver and kidneys are degenerated and pigmented; the brain and many other organs may be gray from pigment. *Pernicious forms* may show masses of parasites in the capillaries of the organs that give the prominent symptoms. Secondary anemia becomes marked in all cases. *Malarial cachexia* has severe anemia, a very large and hard spleen, a large liver, and pigment in these organs and throughout the abdomen.

Symptoms.—*Intermittent forms* have a paroxysm showing three stages: (1) The *chill*, which lasts fifteen minutes or longer, often occurs in the forenoon, is occasionally absent in adults, and often absent in children. (2) The *hot stage*, which is a marked sthenic fever. The temperature is 104°–107° F. within an hour of the chill, and begins to fall an hour or two later. This stage lasts two to eight hours. (3) *Sweating*, usually profuse, lasts half an hour or two hours. With it the patient may sleep, and afterward awake feeling quite well and with a subnormal temperature. The actual pyrexia lasts ten or twelve hours. *Course:* There is a paroxysm every forty-eight hours in the tertian form; every seventy-two hours in the quartan; and every day in the quotidian, with two broods of tertian or three broods of quartan organisms. If untreated, they may become severer, and begin an hour or two ahead of time (anticipating chills); usually they grow less severe, and may be an hour or two late (postponing chills); after a fortnight or so the paroxysms cease, but may recur after weeks or months. During a paroxysm the spleen enlarges, and in the second or third paroxysm is palpable; it recedes a little in the intermission; after several paroxysms it may be felt for a few weeks. The blood contains the organisms, the red cells and hemoglobin grow less after every paroxysm, there are pigmented leukocytes, a leukopenia and a relative lymphocytosis of large lymphocytes. There is a gradual loss of weight and strength, and the symptoms of anemia develop. Herpes of the lips and nose is common.

The æstivo-autumnal form.—The temperature may begin with one or more intermittent paroxysms at intervals of one to three days, which are apt to begin and end more slowly; to last twenty hours or more, and to anticipate, so that they finally overlap and the temperature becomes remittent or even continuous for a week or two; then it falls by lysis. The highest point may be 102°–105° F. General symptoms of fever follow the temperature, but chills and sweats are often absent. The typhoid state may develop. The spleen is large and there may be slight jaundice. Anemia is marked. The blood contains small rings, and later crescents also. *Course:* The patient may recover, but have attacks at intervals for some years, or may develop cachexia or pernicious forms.

Pernicious malaria is almost always æstivo-autumnal, but sometimes tertian. It is rare except in tropical and subtropical places. Many types are described: (*a*) *Algid type:* There is collapse with cold and clammy, pale or cyanotic skin, great abdominal pain, vomiting, perhaps hematemesis, dysenteric or choleraic stools, a very weak heart, a normal or subnormal temperature, and a clear mind. Then coma develops with death in a day. Possibly recovery with profuse sweating may follow. (*b*) *Comatose type:* In remittent fever or instead of a paroxysm there is sudden coma, or acute delirium developing into coma, with a temperature of 106°–108° F., extreme prostration, and death in a few hours or a day. There may be recovery from one attack, but hardly from a second. (*c*) *Hemoglobinuric and hematuric forms:* With a paroxysm the urine may be diminished and contain hemoglobin, albumin, and casts, or in the worst cases blood. There may be bleeding from all the mucous membranes and into the skin, with vomiting, diarrhea, jaundice, delirium, and death in coma or convulsions (perhaps uremic) in a few hours or a day or two. Severe cases rarely recover. There are, however, cases of ordinary paroxysms with hemoglobinuria that usually recover. "Black-water fever" is variously ascribed to pernicious malaria, to quinin, or to a wholly different infection.

Malarial cachexia, in malarial districts, may follow acute attacks or begin insidiously. The symptoms are those of intense anemia, usually of the secondary type, sometimes of a pernicious type, and an enlarged spleen. In malarial districts the patients die of asthenia or intercurrent disease in a year or two. They may recover in a non-malarial district, but the spleen remains large. There may be no parasites in the blood.

The **diagnosis** of malaria is based on finding the organisms in the blood, and is helped by the temperature-curve and the large spleen.

Simple intermittents recover perfectly with treatment. Without it they usually get well, but have recurrences. Æstivo-autumnal malarial cases usually recover with treatment. Without it they often recover, but some cases die of asthenia, in the typhoid state, or of pernicious malaria. *Pernicious cases* sometimes recover with treatment, seldom without it. *Malarial cachexia* is curable in a suitable climate, but hardly in a malarious district.

Treatment.—*Prophylactic:* Drain all pools and marshes. In the mosquito season spread crude petroleum every two weeks over bodies of water. If this is not feasible, stock the bodies of water with fish that eat the larvæ. Screen all openings in houses, especially at night. Kill all mosquitoes in the house every day. Protect by leggings, gloves, and veils the exposed parts of the body. If all *Anopheles* can not be gotten rid of, isolate all malarial patients by mosquito screens around the bed. In a malarial district administer quinin, gr. x daily, to those exposed, treat assiduously for several weeks all who harbor the organism, and renew the quinin in the mosquito season for two or three years. *The attack: Intermittents:* Quinin gr. xx daily, four to six hours before the time for a paroxysm or in divided doses. *Æstivo-autumnal:* Enough quinin to cinchonize the patient. *Pernicious forms:* Bimuriate of quinin and urea, or bimuriate or hydrobromate of quinin, gr. v–xv or more hypodermically, repeated in two hours if the symptoms do not abate; and followed by a solution of quinin by mouth. In all forms give quinin in diminishing doses for several weeks after the fever has disappeared. *Malarial cachexia* and the *chronic cases* cannot be successfully treated in malarial districts.

QUESTIONS.

Describe the asexual cycle of the æstivo-autumnal parasite; the sexual forms in man, and the cycle in the mosquito.

Describe the other forms of the parasite.

Give the other points in the etiology of malaria.

Describe a paroxysm and the course of intermittent malarial fevers.

Give the symptoms of the æstivo-autumnal type.

Give the varieties of pernicious malaria and their symptoms.

Describe malarial cachexia.

What are the preventive treatment and the curative treatment?

MALTA FEVER.

The specific **cause** is Micrococcus melitensis, which is found in the spleen and often in the urine. It is not directly contagious; is endemic in Malta and other places; occurs in the Philippines, China, and elsewhere.

There are no characteristic **lesions.**

Symptoms.—The *incubation period* is about a week. There is a fever (102°–104° F.), rather continuous, ending by lysis after one to three weeks. Then after an intermission of a few days the fever may assume (1) the *undulatory type,* in which there is a series of febrile periods, lasting about two weeks, with remissions of a few weeks, the whole continuing three to six months or longer. Anemia, an enlarged, tender spleen, and loss of weight and strength are present. (2) The temperature may be *intermittent;* in the relapse the fever has daily intermissions; after two or more weeks the fever disappears for a few weeks and then returns. Convalescence is slow. About 2 per cent. die; but the disease makes the patient an invalid for several months, There is a *diagnostic agglutinative reaction* with the micrococcus and the patient's serum.

Treatment is on general lines, but recovery is hastened by removal to another place.

QUESTION.

Give the etiology, the temperature-curve, and the diagnostic points of Malta fever.

BERI-BERI.

Etiology.—This is an endemic disease of the tropics of unknown etiology. It may occur in epidemics and has been found in America. It is more common in young mále adults, and is not contagious.

The **lesion** is a multiple neuritis of the vagus, the phrenic, and the nerves of the limbs and face.

Symptoms.—The *incubation period* is from one to several months. There are several clinical types: (1) *Rudimentary form* involves the nerves of the limbs and the heart; with paresthesias, tenderness and paralysis of the muscles, and slight edema; also a rapid, weak heart action, palpitation, and dyspnea. Recovery follows after a few days or many months. Recurrences are common. (2) *Atrophic form:* The symptoms in the limbs are more marked, with atrophy and reaction of degeneration. The face also may be attacked. This lasts for months, but may end in recovery. (3) *Dropsical form:* There is much dropsy of the connective tissues and serous sacs, but no albuminuria. Heart symptoms are present; many cases die within a month, others may recover. (4) *Acute pernicious form:* This may start like the rudimentary form, but the heart symptoms become severe and are fatal. The general mortality varies from 2 to 60 per cent. in different epidemics.

Treatment.—Good air and sanitation, nitrogenous food, salicylates in large doses.

QUESTION.

Give the etiology, lesion, and symptoms of beri-beri.

ANTHRAX.

Etiology.—Bacillus anthracis is a large bacillus that forms spores which resist boiling for three minutes. The vegetative forms are killed more readily. The bacillus is found in the local lesions, and after three or four days in the blood. Infection is usually by inoculation, sometimes from food or water, rarely by inhalation. This is a disease of sheep and cattle, but is found in persons who handle animals, their hides, wool, or carcasses. The injection of attenuated cultures confers immunity, at least in lower animals.

Lesions.—(1) *Malignant pustule:* At the site of inoculation, after a day or so, a firm red papule appears; on the second day it becomes a vesicle with bloody contents; after another day this ruptures and forms a dark scab. Around this is a ring of miliary vesicles with clear contents and an area of inflammatory edema that may be rather large. There may be lymphangitis and inflamed lymph-nodes. The inflamed tissues contain many bacilli. Healing occurs by sloughing and granulation. (2) *Intestinal mycosis:* In the stomach and intestine are brown patches infiltrated with pus at the center, and around this with blood. The bacilli abound in the lesion. The gut contains flakes of fibrin, the spleen and lymph-nodes are swollen, there is serum in the peritoneal cavity, and the lungs may show small hemorrhages, edema, or pleurisy. In all, after a few days, the blood contains anthrax bacilli.

Symptoms.—*Incubation period:* A few hours to three days. (*a*) *External anthrax:* (1) *Malignant pustule:* The pustule develops, as described above, with itching and burning. The general symptoms are a high fever (105° F.), and after three or four days the typhoid state, and death within a week, or gradual recovery. Pustules on the head and neck are more often fatal than those on the limbs. (2) *Malignant anthrax edema:* There is a rapidly spreading, hard edema, and sometimes necrosis and gangrene. It is most common in the eyelids, neck, or arms. There is intense toxemia, and all the cases die in a few days. (*b*) *Internal anthrax:* (1) *Intestinal mycosis:* There is an abrupt onset, with vomiting, purging, bleeding into the skin and mucous membrane, fever

(106° F.), intense toxemia, and death in a few days. (2) *Respiratory or pulmonary form* (wool-sorters' disease): There are cough, dyspnea, pain in the chest, suffocation. The sputum contains anthrax bacilli and blood. There is intense toxemia, with death in a day or week.

Treatment.—*Preventive:* Infected animals must be isolated and their carcasses burned or buried more than six feet deep. Exposed animals may be immunized. *Malignant pustule* should be excised early.

QUESTIONS.

Give the etiology of anthrax.

Give the lesions and symptoms of internal anthrax, of external anthrax.

HYDROPHOBIA.

Etiology.—Specific cause is not known, but it seems to exist in the central nervous system and the secretions of diseased animals. It is transmitted by inoculation with the bite of an infected dog, wolf, cat, cattle, horse or skunk.

Immunity.—Virus is intensified by successive inoculation of a series of rabbits with emulsions of the spinal cords. When the cord of a rabbit that died after inoculation with this strongest virus is dried, it is found to become progressively less virulent. If mulsions of such cords are inoculated in increasing strengths until the strongest is reached, the animal thus treated is then immune to subsequent bites of mad animals and the immunity seems to last for at least two years. In man, if the inoculations are begun within five days after the bite, immunity is almost surely established. If begun later the result is less certain.

Lesions.—The wound of inoculation heals normally. At death there are lesions of the central nervous system, especially in the bulb—congestion, groups of round cells, minute hemorrhages, and degenerations of the nerve-cells.

Symptoms.—The *incubation period* averages six weeks, but may be several months. The *prodromal symptoms* are itching at the site of the bite and great apprehension. (1) *The stage of excitement:* There are irritative nervous symptoms, such as great apprehension, excitement, intermittent delirium which is often maniacal, tonic spasm of the pharynx, of the respiratory muscles, and of the whole body, hyperesthesia of the general and special senses. General tonic convulsions may be induced by attempts at swallowing, a draught of air, a noise, etc. In these convulsions cyanosis is marked and there may be fatal asphyxia. There is moderate fever. The urine contains albumin or blood. (2) *Paralytic stage:* There are symptoms of nervous paralysis involving the muscles and sensation. There may be coma, and death follows from exhaustion or asphyxia in three or four days in every case. Of those bitten, from 2 to 80 per cent. get the disease. Bites on the face and neck are most dangerous, those through clothing the least dangerous.

Treatment.—*Preventive:* Immediate cauterization of the wound, and inauguration of the immunizing treatment.

QUESTIONS.

Discuss the etiology of rabies, and immunization.
Give the symptoms.
What is the prognosis of the bite of a mad dog?
What is the prognosis of hydrophobia?
What is the treatment of a suspicious bite?

TETANUS.

Etiology.—The specific cause is Bacillus tetani, which is anaërobic and forms spores. It is found in the body at the point of infection only. It occurs in the soil, in manure, and in the intestines of herbivora. It enters by inoculation. Antitetanic serum gives immunity if injected within a day or two of infection. Tetanus is endemic in certain districts.

There is no characteristic **lesion** except the bacilli at the site of infection.

Symptoms.—The *incubation period* is about two weeks. The *onset* is gradual. The striking symptoms are tonic spasm of all the muscles, with remissions (but not intermissions) and extremely painful exacerbations, which are usually of reflex origin. This begins as trismus and a stiff neck; it spreads gradually to the trunk and legs, and is least marked in the arms. The risus sardonicus is present. In exacerbations there may be opisthotonos, some respiratory spasm, and asphyxia. These exacerbations are induced by any slight stimulus, and last a few seconds or a minute or more. If the patient improves, the muscles relax in the remission, the spasms become less frequent, less severe, and are less easily induced. Fever is variable. The mind is usually clear. Death is due to asphyxia, exhaustion, or heart failure.

Course and Varieties.—*Idiopathic cases* are the least fatal; there is no discoverable wound of inoculation. *Acute cases* last from a day to a week, and most die. *Chronic cases* have less intense symptoms, last two or three weeks, and are less fatal. *Tetanus neonatorum* is due to infection at the navel, and is fatal. The general mortality of traumatic tetanus is about 80 per cent.

Treatment.—*Preventive:* After a suspicious wound the person should be immunized at once by the injection of 500 units of antitetanic serum. Suspicious wounds should be carefully cleaned and drained freely. *When the symptoms develop,* antitoxin is used, but it is not very successful. The wound of inoculation should be opened and cauterized, or excised. During the attack all stimuli, such as strong light, sound, and mechanical irritation, should be avoided. Feed by the rectum or with the stomach-tube, using chloroform to avert the spasms if necessary. For the spasms chloroform inhalation is most effective. Morphin, chloral, bromids, physostigma, or carbolic acid are given. Symptomatic treatment is needed.

QUESTIONS.

Give the etiology, incubation period, symptoms, prognosis, and the treatment of tetanus.

Discuss antitetanic serum.

ACTINOMYCOSIS.

Etiology.—The specific cause is Streptothrix actinomyces, or ray fungus, which is found in the lesion and discharges. It can be grown in cultures, and is supposed to enter by inoculation. This is a rare disease.

Lesions.—There is a miliary mass composed of small round cells, epithelioid cells, giant cells, and the actinomyces. The ray fungus is a yellowish granule, 1 or 2 mm. in diameter, made up of cocci, with radiating branches clubbed at the ends. These miliary nodules occur in groups, and among and around them is a chronic productive inflammation, forming large hard tumors which necrose and form abscesses. This lesion is found in any part of the body and spreads by extension or metastasis. There may be an associated exudative inflammation of surrounding tissue.

Symptoms.—There are the symptoms of general sepsis, which may even be fatal, of local tumors, abscesses, and suppuration, and sometimes of pyemia with metastatic foci. The first lesions noted may be in the alimentary canal, the liver, spleen, lungs, or skin.

The **diagnosis** is made by finding the ray fungi in the discharges, such as the saliva, sputum, stools, or pus, depending upon the situation of the lesion. As a rule there is some leukocytosis. The disease lasts three months to a year or longer, and death usually follows from chronic sepsis.

The **treatment** is that of pyemia. Potassium iodid, ʒj a day, is recommended.

QUESTIONS.

What is the etiology of actinomycosis?
Describe the lesion and give the symptoms.

SYPHILIS.

Etiology.—The specific cause has not been demonstrated. It seems to exist in the primary and secondary lesions and the discharges from them, and in the blood in the secondary stage. Outside the body it retains its virulence for a time. It enters the body by inoculation, usually in sexual intercourse, but not rarely in other ways, as in kissing, a scratch on the hand or nipple, etc. It may be congenital from a syphilitic father with or without infection of the mother, or from a syphilitic mother, or from both parents. If the mother is infected after conception, the child may be infected through the placenta. Immunity follows an attack. If a nonsyphilitic woman bear a syphilitic child, she is immune, at least from infection by her child (Colles' law). Males may transmit the disease until the end of the secondary stage, seldom after three years. Less is known about this point in women.

Lesions.—They are very like those of tuberculosis. The chancre, the primary lesion, is composed of small round cells, epithelioid cells, and perhaps giant cells. It tends to necrose, to ulcerate, and to cicatrize. The gumma, a tertiary lesion, is a mass of round cells and epithelioid cells, and it tends to necrose and become cheesy at the center; around this in time a translucent zone of firm fibrous tissue is formed. If the necrosis reaches a surface, a deep, punched-out ulcer with overhanging edges and a necrotic wall is formed. In healing a scar forms. There are other lesions also in the skin and mucous membrane and in the lymph-nodes. There is a tendency to chronic productive inflammations in after-years, especially in the arteries, and to certain nervous diseases. There are secondary changes in many organs.

Symptoms.—*The acquired form:* The *incubation period* is about three weeks. Then the chancre (*primary stage*) appears, and after a few months leaves a scar; the related lymph-nodes are large, hard, and painless. The *secondary stage* begins about six weeks after the chancre appears, lasts several months or a year or two, but with treatment usually only a few weeks. The rashes are usually symmetrical, superficial, without itching, and often leave copper-colored pigmentation. They may be roseolar, papular, squamous, or even pustular. At mucocutaneous junctions papillæ hypertrophy and form *syphilitic warts.* If these run together and are kept moist, they form *broad condylomata.* In the mouth, nose, vulva, or anus the tissue may be swollen and grayish, forming a *mucous patch.* In this stage there is some fever, and there may be loss of weight, anemia, temporary loss of hair, onychia, iritis, other eye inflammations, deafness, synovitis, or nocturnal pains in the bones. Usually the symptoms intermit for a time, and then the *tertiary stage* starts, three or four years after infection. It is, however, often absent, especially after treatment. The tertiary skin lesions are asymmetrical and tend to scar. Rupia is a form with deep ulceration and scaly crusts ; it is called a late secondary symptom by some, and may transmit the disease. Anywhere in the body gummata or gummatous tissue may appear.

Syphilis of the Various Organs.—In the *rectum* there may be a stricture just above the internal sphincter from a scar of a gumma in the submucous coat. It is more common in women. In the *liver* gummata form swellings and later depressed scars. Clinically there is a large, very irregular liver, with fair health and a history of syphilis ; there may be ascites and jaundice. The *heart* rarely contains gummata. There is a tendency to aortic endocarditis, arteriosclerosis, or gummata in the outer coat of the arteries. In the *respiratory tract* there may be necrosis of the bones of the nose, with saddle nose or other deformity. Gummata with ulcers may form on the epiglottis or vocal cords, causing hoarseness, aphonia, cough, or edema of the glottis. Later, there may be cicatricial stenosis and deformity. Syphilis of the *lungs* is rare. In the *central nervous system* there may be gummata in the pia or the blood-vessels, thickening of the pia, or arteritis. As a consequence of these lesions there may be areas of softening of the brain or hemorrhage. The usual site is about the middle cerebral artery. They may cause also epileptiform attacks preceded by headache, dizziness, and excitement, and followed for days by torpor or the symptoms of brain tumor, or symptoms like dementia paralytica. In the *spinal cord* there may be symptoms of tumor or myelitis. *Genito-urinary organs:* In the testis there may be gummata which are hard and painless, and may suppurate, but usually resolve without suppuration ; there may be interstitial orchitis with atrophy. In women there are miscarriages. The *bones* are liable to periosteal nodes, especially on the shins, forming a hard, permanent swelling with nocturnal pain. The bones of the nose are especially apt to necrose. There may be a *chronic synovitis* or gummata around the joints.

Parasyphilitic affections are not strictly syphilis, but are more common in syphilitics. They include locomotor ataxia, dementia paralytica, epilepsy, chronic productive inflammation, especially in the arteries (including aneurism), and amyloid disease.

Congenital Syphilis.—Symptoms at birth are rare, except with abortion and stillbirth; there may be bullæ on the hands and feet, nasal obstruction, fissured lips, enlarged liver and spleen, periostitis, epiphysitis, bleeding in the skin and elsewhere, emaciation, and death in a few days. Usually the child looks healthy at birth, but in the second month develops the symptoms. There are rashes appearing first on the nates, usually erythematous, sometimes papular, eczematous or pustular; fissures and ulcers at mucocutaneous junctions, snuffles, seropurulent or bloody discharge from the nose, alopecia, onychia, enlarged lymph-nodes and spleen, and otitis media. The skull shows prominent frontal eminences, thickening around the anterior fontanel, and craniotabes. The liver is large, yellow, and tough on section: it shows areas of small cells and later radiating strands of fibrous tissue (diffuse syphilitic hepatitis). During this stage the child may infect others. The symptoms disappear as a rule; but if the child lives, there are late symptoms in the second decade, such as Hutchinson teeth (the upper central, permanent incisors are small, conical, and notched at the edge); keratitis, iritis, deafness without aural discharge, symmetrical and painless periosteal nodes on the long bones, enlargement of the liver or spleen, and atrophy of the testes. Gummata, amyloid disease, and other tertiaries are rare.

Diagnosis.—In the secondary stage this is usually easy. In the tertiary stage one must get a history of the chancre (this is especially doubtful in women), of rashes, sore mouth or perineum, alopecia or miscarriages. The patient may have a saddle nose, copper-colored scars on the shins, nodes on the bones, atrophied testes, etc. The therapeutic test with potassium iodid is often valuable.

Prognosis.—*Congenital cases* with symptoms at birth always die; other cases often die with poor treatment, seldom with good treatment. *Acquired cases:* With good treatment they may have only a few secondaries; but there are malignant cases that die in a year or two in spite of treatment. Treatment does not prevent parasyphilis.

Treatment.—*Prophylaxis* involves the solution of the sexual problem. During the contagious stage all fomites and discharges should be disinfected and contact with syphilitics should be avoided. *Curative treatment:* Careful hygiene, baths, feeding, and temperance are important. During the primary and secondary stages mercury may be given by inunction; by mouth (the iodids or gray powder); by fumigation (calomel); or by deep injection, usually into the gluteal muscles, of the bichlorid gr. $\frac{1}{8}$ once a week. This treatment should be kept up with intermissions for at least two years. For tertiary symptoms sodium or potassium iodid is given in increasing doses to the limit of toleration, for several months. In the intermediate period the symptoms often yield best to combinations of mercury and iodin. Usually tonic treatment with iron, etc., is indicated in most cases. Parasyphilitic conditions do not yield to these remedies.

QUESTIONS.

Give the etiology of syphilis.
What is Colles' law?
Discuss the hereditary transmission of syphilis.
What are the lesions of the primary stage and of the tertiary stage?
Give the symptoms.
What is meant by parasyphilis?
Give the treatment of syphilis.

TUBERCULOSIS.

Etiology.—The specific cause is Bacillus tuberculosis. It occurs in the lesions and the discharges from them (sputum, pus, stools, etc.). Outside the body it retains its virulence for an indefinite time, and is found in the dust of rooms occupied by consumptives, having come from sputum either dried, or atomized by talking or laughing. It is found in tuberculosis in some lower animals, and may get in milk or on meat. It enters the body with the breath, or in contaminated milk or other food, rarely by inoculation or, in the fetus; from the placenta. The bacillus is killed when dry by direct sunlight in four to six hours; when wet by boiling for three or four minutes, by 160° F. in fifteen minutes, by carbolic acid and mercury bichlorid in the ordinary concentration. The necessary factors for infection are the bacillus and individual susceptibility. (1) Contact with the bacillus is favored by intimate contact with tuberculous patients, at home, in hospitals, prisons, or asylums; by residence in rooms or houses that have been occupied by patients and not thoroughly disinfected; by handling fomites, such as handkerchiefs and bed linen, or tuberculous tissues, especially in the post-mortem room. (2) Individual susceptibility exists at all ages in both sexes, rather more in negroes and in Indians, it is heightened by bad ventilation, dampness, probably by low altitudes, and especially by a lack of robust health or by actual disease, such as bronchial catarrh, pneumonoconiosis, certain infectious diseases, especially pertussis, measles, influenza, or variola; by diabetes, chronic disease of the heart, arteries, or kidneys, by enterocolitis, by trauma of the head, chest, or joints. Heredity predisposes by conferring a general lack of vigor and the probability of contact with tuberculous patients. Inheritance of the actual disease (tuberculosis of the fetus) is extremely rare. Bovine tuberculosis is usually thought to be the same disease transmissible in milk, especially from cows with diseased udders; or by meat contaminated in slaughtering.

Lesions.—*The tubercle tissue:* (*a*) A miliary tubercle is a yellow, gray, or white mass, from microscopic size to half a centimeter in diameter, composed of giant cells, small round cells (lymphocytes), and epithelioid cells (derived from connective-tissue cells and the endothelium of the capillaries), with tubercle bacilli in and among the cells. These constituents occur in varying proportions, and all of them are not always present. (*b*) Diffuse tubercle tissue has a similar structure, but occurs diffusely in masses of all sizes. All tubercle tissue contains few or no blood-vessels, and tends to degenerate and undergo coagulation-necrosis, forming a granular or cheesy mass. The necrosis begins at the center and spreads rapidly or slowly; later the cheesy material may be calcified. The tubercles may be solitary or aggregated in groups, and there may or may not be areas of diffuse tubercle tissue among them. These lesions may appear in any part of the body. In the surrounding tissue there is usually a zone of inflammation. This may be productive, and sometimes results in the encapsulation of the cheesy mass within fibrous tissue (healed tubercle). Or there may be exudative inflammation. There may be secondary infection with pyogenic cocci, and suppuration. Possibly the tubercle bacilli alone may cause suppuration. The necrosis may erode a blood-vessel and result in the rapid and general dissemination of the bacilli, with consequent general infection. The inoculation of dead bacilli causes characteristic tubercles. *The site of the lesion:* In fatal cases this is seldom limited to one organ. In adults the lungs and some lymph-nodes are almost always involved; the other sites are the larynx, intestine, peritoneum, genito-urinary organs, brain, bones, joints, and skin. In children the most common sites are lymph-nodes, intestine and peritoneum, bones and joints, and meninges.

Prognosis.—Very many cases recover. Tuberculosis is said to cause one-seventh of all deaths, but in many cities the mortality has been lessened by one-third or one-half during the past generation.

Specific Diagnosis.—(1) Tuberculin injected hypodermically is followed by a rise of temperature to 101° F. within one or two days, if the patient has tuberculosis. This is very useful in afebrile states, but not infallible. (2) Demonstration of the tubercle bacilli in sputum, urine, or other discharges. A smear of the material on a slide is dried in the air, fixed in a flame, stained one to three minutes in steaming carbol-fuchsin solution, decolorized by acid alcohol, and counterstained with methylene-blue. The tubercle bacilli only are stained red. (3) Inoculation of suspected matter into a guinea-pig's peritoneal cavity or subcutaneous tissue. Within two weeks the lesions of tuberculosis may be demonstrated by microscopic examination of the peritoneum or the related lymph-nodes. This is more reliable than the search for the bacilli.

The Blood in Tuberculosis.—Without secondary infection the red cells and hemoglobin are surprisingly normal, the leukocytes normal or diminished (except meningeal tuberculosis). With secondary infection by pyogenic germs there is a leukocytosis. Secondary infection is common in the lungs with cavity formation, in the genito-urinary tract, lymph-nodes or tubercular abscesses that have been opened or have ruptured on the surface.

QUESTIONS.

Give the etiology, general lesions, and prognosis of tuberculosis in general.

Describe the methods of specific diagnosis, including the staining of the bacilli.

Discuss the blood in tuberculosis.

ACUTE GENERAL MILIARY TUBERCULOSIS.

Etiology.—This is secondary to previous tuberculosis either active or latent, such as infected bronchial or mesenteric nodes, or as the terminal event in any tubercular process.

Lesions.—A tubercular focus may break into blood-vessels or the thoracic duct, and many bacilli soon lodge in many places. The lesions (p. 106) are found scattered in the lungs, meninges, liver, spleen, and elsewhere. The necrosis and cavity formation are usually confined to small scattered foci.

Symptoms.—With a history of some existing or previous tuberculosis or of measles, pertussis, or other infectious disease, the patient becomes ill. Three groups of cases are usually described: (1) *Typhoid type:* The local symptoms are slight, but there is a general toxemia that resembles typhoid. The skin, however, shows no roseola that is typical in site, time, and duration, although there may be irregular rashes and perhaps herpes. Usually there is less tympanites, hemorrhage is rare, there is no perforation, but the spleen may be slightly enlarged, the pulse is faster, leukocytosis is rare, the breathing more rapid, there is more bronchitis, often cyanosis, the temperature lacks the typical rise, and is more irregular. The specific diagnostic points of typhoid are absent; those of tuberculosis may be present. There may be choroid tubercles. The disease ends in death after four to eight weeks from exhaustion, but it often merges into one of the other types, or some local tuberculosis may become evident. (2) *Pulmonary type:* After a slight cough or in the course of a chronic pulmonary tuberculosis or an infectious disease, such as measles or pertussis, the patient becomes acutely ill with the symptoms of an acute pulmonary tuberculosis, usually of the bronchopneumonic type (p. 112), and dies in two weeks or a few months. Other foci of tuberculosis may become apparent, especially in the meninges. (3) The *meningeal type* has been described on p. 60.

QUESTIONS.

Give the special points in the etiology and lesions of acute general miliary tuberculosis.

What are the common clinical varieties?

Give the differential diagnosis between the typhoid type and typhoid, between the pulmonary type and bronchopneumonia, between the meningeal type and other meningitis.

TUBERCULOSIS OF THE LYMPHATIC SYSYEM.

Lymph-nodes are often involved at any age, but usually in children. Catarrhs strongly predispose. *Lesions:* The lymph-nodes are much swollen, and show hyperplasia and the tuberculous lesions. They tend to run a very chronic course, to soften and rupture externally, forming suppurating sinuses, which become infected with pyogenic cocci. The sinuses persist for months, but heal finally with an irregular scar. The node may become quiescent, but afterward be the starting-point of active tuberculosis elsewhere in the body.

Symptoms.—(1) *General involvement of lymph-nodes:* The cervical, axillary, thoracic, and abdominal nodes swell and cause great deformity. After a few months or years the patient dies of exhaustion or of acute tuberculosis. This form is rare and resembles Hodgkin's disease. (2) The *cervical glands* on one or both sides of the neck are very often involved, usually from catarrh of the throat or large tonsils. The nodes often suppurate. There may be some loss of flesh and strength, usually fever is absent. Tuberculosis of the pleura or lung may follow. (3) The *bronchial nodes* are involved secondarily to bronchitis or pneumonia, especially with measles and pertussis. Local symptoms are usually absent, but these nodes are often the starting-point of a general tuberculosis in later years. (4) The *abdominal nodes* may be infected after enteritis. The nodes in the mesentery and behind the peritoneum may form large masses. There are a distended abdomen, diarrhea, emaciation, irregular fever. The masses may sometimes be felt. Often there is tubercular peritonitis or generalized tuberculosis.

The **serous sacs** are usually infected by extension from contiguous structures. They are seldom the primary focus.

Lesions.—The *tubercular part* may be (*a*) scattered miliary tubercles with nothing else; (*b*) diffuse tubercle-tissue in plates which form cheesy masses. The *associated inflammation* may be (*a*) exudative with fibrin and serum, often bloody, or pus; (*b*) productive inflammation which may encapsulate old tubercles, or be progressive and form extensive adhesions, or may lead to sacculation of pus. These lesions are combined in various ways. The *course* may be acute or chronic. One or more sacs may be involved, or there may be lesions in other organs, so the clinical picture is most varied. Sometimes there are latent cases without noticeable symptoms.

General Serous Membrane Tuberculosis.—The serous sacs are involved together or in succession. (1) *Acute cases*: There are miliary tubercles and an exudate of serum or pus. (2) More *chronic cases* have tubercles, diffuse tubercle tissue with cheesy plates, and an exudate, usually of pus. (3) The *productive type* has hard fibroid tubercles imbedded in thickened membranes.

Tubercular pleurisy may begin acutely, but usually becomes chronic. The exudate may be pus or serum. Chronic adhesive pleurisy is also seen. These cases resemble ordinary pleurisy, but are more persistent.

Pericardium: Latent cases are common. There may be acute cases with exudates of serum or pus; or there may be adherent pericardium.

Peritoneum: (1) *Latent cases* are common. (2) The *acute cases* become very ill at once, and resemble ordinary acute peritonitis with ascites. (3) There are *less acute cases* with ascites which may be sacculated. The liquid may be serous or purulent. (4) In *chronic cases* the intestine and omentum form irregular masses of doughy feel. There are loss of appetite, indigestion, diarrhea, abdominal pain, tympanites, a varying temperature and progressive emaciation, also the physical signs of the masses of intestines and omentum and of exudates which may be sacculated. Pigmentation of the skin is common. Some cases recover. The diagnosis is based on the physical signs of the tumor masses and the ascites, on the absence of leukocytosis, upon finding a primary focus of tuberculosis elsewhere, and upon the tuberculin test when feasible.

QUESTIONS.

Describe the lesions in tuberculous lymph-nodes and give the special points in the etiology.

Give the symptoms of general lymph-node tuberculosis.

Give the symptoms of tuberculosis of the cervical nodes, of the abdominal nodes.

What are the causes and the possible sequels of tuberculosis of the bronchial nodes?

Describe the general lesions of tuberculosis of the serous sacs.

Give the varieties of general serous membrane tuberculosis, of tuberculosis of the pleura, of the pericardium.

Describe the lesions of tuberculous peritonitis, give the symptoms and detail the physical signs.

PULMONARY TUBERCULOSIS.

The bacilli may be carried to the lungs in the blood- or lymph-vessels, or may be inhaled. The lesion corresponds to tuberculous lesions elsewhere. The *miliary tubercles* in acute cases are softer and very cellular; in slower cases they may be cheesy at the center, and later be replaced by firm, fibrous nodules. *Diffuse tubercle-tissue* sometimes forms large masses which may become cheesy, and in that case usually liquefy and are discharged through a bronchus, leaving a cavity. The *associated exudative inflammation* causes consolidation with the air-spaces and small bronchi filled with fibrin, pus, and epithelium. This may involve one lobe like a lobar pneumonia, or be in scattered smaller areas, like a bronchopneumonia. The exudative part of the inflammation may resolve. *Necrosis* beginning in the tubercles or tubercle tissue spreads to the intervening parts of the lung and forms cavities of any size. In the less acute cases attempts at *healing* by the formation of fibrous tissue take place. This may result in the encapsulation of the focus, often with calcification and complete arrest of the process, leaving a puckered scar; a small cavity may perhaps be obliterated in this way; these scars are often found at autopsy. There may be *progressive interstitial pneumonia*, with contraction of the lung. There is more or less *bronchitis*, either acute or chronic; in the chronic form *bronchiectases* may form with smooth and clean or with suppurating walls. There are also tuberculous *bronchial nodes*. There may be fibrinous or fibrous *pleurisy*, and in other parts of the lung *emphysema*. *Cavities* may be large or small. (1) They may have soft, shaggy, necrotic walls. Sometimes an artery stretches across such a cavity and often shows little aneurysms. (2) The walls may be rather smooth granulation-tissue. (3) The cavity may be bronchiectatic. The tuberculosis usually starts near one apex and spreads downward. The other lung is very often attacked. One lung may be extensively consolidated, while other parts may contain only tubercles. Tubercle bacilli appear in the sputum when softening takes place. Much of the necrosis, suppuration, and exudative inflammation is due to secondary infection with streptococci. Tuberculosis in other organs is very common.

ACUTE PNEUMONIC PHTHISIS.

There is (1) a *lobar pneumonic type* that begins like lobar pneumonia, but may have an early hemoptysis. At the end of the first week there is no crisis, but the disease continues, night-sweats come on, there are cyanosis, a hectic temperature, a faster pulse, an abundance of moist râles, greenish and mucopurulent sputum containing tubercle bacilli. After a month there may be the signs and symptoms of a cavity with hemoptysis. The mind is clear and hopeful. Emaciation is marked. Death may follow in two weeks or after a few months. Occasionally the exudative pneumonia resolves and the case becomes chronic phthisis. (2) The *broncho-pneumonic* type is more common *in children*, especially after measles, pertussis, etc. There are the symptoms of a more or less typical bronchopneumonia which may be rapidly fatal, or may abate and become chronic. In *adults* the onset is less intense than that of the lobar form; later chills, night-sweats, a remittent temperature, the typhoid state, and emaciation develop. The lung symptoms are those of bronchopneumonia. Hemoptysis is common. There are the physical signs, first of general bronchitis, with or without pleurisy; later of spreading areas of consolidation, which may run together and assume the lobar type. Cavities may develop. Death in two to eight weeks is usual, but some cases become chronic. If tubercle bacilli are found in the sputum, a positive diagnosis may be made.

CHRONIC PHTHISIS.

This is the most common form of lung tuberculosis. It follows acute phthisis or is chronic from the start. Some cases are *latent* until the lesion is well advanced.

Any one or more of the following **symptoms** may appear first, but later others are added: Dyspepsia; diarrhea; a persistently rapid heart; anemia and its symptoms; respiratory symptoms—hoarseness and pain in the larynx, a tendency to bronchitis, pain over the lungs, hemoptysis, accelerated breathing, pleurisy; a slight afternoon temperature, or an intermittent fever, with chills and sweats; enlarging lymph-nodes of the neck; inexplicable loss of weight. When the disease is well established, there are anorexia, often nausea, vomiting, and attacks of diarrhea; a rapid, soft pulse; anemia; a cough with mucopurulent sputum that contains tubercle bacilli; hemoptysis, often large and repeated, or followed by an exacerbation of the process; perhaps pain in the lung; a fever, remittent or intermittent; sweating, especially during the night; the hopeful mind; and progressive emaciation and weakness. There are remissions and exacerbations lasting weeks, but, on the whole, a downward tendency. With *cavities* there are more apt to be chills, sweats, and intermittent fever, hemoptysis, a leukocytosis, more rapid emaciation, and the suggestive physical signs.

Physical Signs.—A long, narrow thorax, with sloping ribs, winged scapulæ, flat chest, funnel breast, or depressed sternum, is suggestive. The earliest signs may be one or more of the following: Retraction and deficient expansion above and below the clavicle on one side. *Palpation:* At one apex there may be deficient expansion and increased fremitus in front or behind. *Percussion:* At one apex there may be tympany or dulness. Auscultation: At one apex there may be feeble, hoarse, cog-wheel or broncho-vesicular breathing; increased vocal resonance; crepitant or subcrepitant râles or the signs of a local bronchitis. These signs may appear in front, behind, or in the axilla, either at the apex or a little below. They are more frequent on the right side. Later, other signs are added to the ones that appeared first and signs of consolidation at an apex become distinct. By this time there are usually some of the earliest signs over the other apex. *Cavities* form later. Their signs often are not distinctive, but are—on *inspection*, more retraction at an apex. On *palpation*, increased fremitus. On *percussion*, dulness, tympany, cracked-pot (with the mouth open) or a surprisingly normal note. On auscultation, cavernous or tubular breathing and voice; pectoriloquy; large, moist, gurgling, râles, often metallic and more abundant on coughing. The signs vary with the amount of liquid in the cavity. Very large cavities give the signs of pneumothorax. The *sputum* with cavities is mucopurulent, opaque, airless, and contains many tubercle bacilli and some elastic fibers; it often is abundant with a paroxysm of coughing, which is apt to be induced by a change of position or exertion.

Other Symptoms.—Diarrhea may be unusually marked with or without tuberculous enteritis. The *heart:* There may be visible pulsation over the second and third left spaces, functional murmurs, or rarely endocarditis. *Respiratory organs:* Tuberculous laryngitis is common; bronchitis may be unusually marked and sometimes is fetid; abscess or gangrene of the lung occurs; attacks of exudative pneumonia and pleurisies are common. *Nervous system:* There may be local tuberculosis of the brain, or neuritis. There may be tuberculosis of any other organ of the body. Pigmentation of the skin, chromophytosis, clubbed fingers, and amyloid disease are common. In the aged the signs are often masked by emphysema and chronic bronchitis. The course is usually slow, and latent cases are common.

The average duration of chronic phthisis is three or four years; death is due to an acute exacerbation, to general tuberculosis or a secondary pneumonia, to asthenia, hemoptysis, or an intercurrent disease.

FIBROID PHTHISIS.

This term is sometimes used for an interstitial pneumonia that is not tubercular. The tubercular cases may follow acute phthisis, tubercular pleurisy, or chronic phthisis. The symptoms are those of interstitial pneumonia, but sometimes the diagnosis may be made by the history, by finding a few tubercle bacilli in the sputum, or by hemoptysis.

QUESTIONS.

Acute pneumonic phthisis: Give the lesions, symptoms, course, and terminations of the lobar pneumonic type, and the differential points from lobar pneumonia.

Give the same for the bronchopneumonic type in children and in adults.

Chronic phthisis: Give the various modes of onset and the first symptoms.

Give the earliest physical signs.

Detail the signs of the ordinary fairly marked case.

Give the signs and other symptoms of cavities.

Give the rational symptoms of chronic phthisis.

Discuss the prognosis.

What is fibroid phthisis?

TUBERCULOSIS OF THE ALIMENTARY TRACT.

The *tonsils* are probably involved rather often, and may be the first point of entrance for the tubercle bacilli. Microscopically they show tubercles. The cervical nodes also are usually tuberculous.

In the *pharynx* adenoids may be tuberculous. There may be a tuberculous ulcer on the pharynx which is very superficial, irregular, with a gray, granulating base, ill-defined edges, and a little grayish secretion that may contain tubercle bacilli.

Intestinal tuberculosis may be primary in a child, or secondary to tuberculosis elsewhere, especially to chronic phthisis. The lesions usually are in the colon, cecum, and ileum, involving the lymphoid tissue chiefly. Necrosis results in ulcers that are round or oval and encircle the gut, with infiltrated and often cheesy edges and base. There are also tuberculous inflammation of the muscular and serous coats and the mesenteric nodes. Chronic cases may heal with scars that may later constrict the gut. An ulcer may perforate and cause general peritonitis. The *symptoms* are constant or intermittent diarrhea, perhaps hemorrhage or perforation, with emaciation and fever. Later there may be a stricture of the gut. There is usually tuberculosis elsewhere. *Ileocecal* cases may look like recurrent appendicitis or cancer of the cecum, with a tumor or ill-defined mass in the right iliac region. It may cause obstruction or a fecal fistula. Tubercle bacilli may be found in the stools with tuberculous enteritis. Fistula in ano is often tuberculous.

Secondary tuberculosis of the liver is common with general and pulmonary tuberculosis, but usually gives no symptoms. The *pancreas* may be involved with tuberculous peritonitis, but usually gives no symptoms.

QUESTIONS.

Discuss tuberculosis of the tonsils.

Describe tuberculous ulcers in the pharynx.

Give the etiology, the lesions, and the symptoms of tuberculosis of the intestine.

TUBERCULOSIS OF THE CENTRAL NERVOUS SYSTEM.

Besides tuberculous meningitis there may be tuberculosis in the cerebellum, the cerebrum, or the pons. There is a nodule of tubercle-tissue surrounded by a translucent zone of connective tissue (solitary tubercle). It is from one-fourth to one inch in diameter, is usually attached to the pia in a sulcus; may press on a venous sinus and cause thrombosis. The symptoms are those of brain tumor, associated with tuberculosis elsewhere. Perhaps the tuberculin test may help in diagnosis.

QUESTION.

Discuss the solitary tubercle of the central nervous system.

GENITO-URINARY TUBERCULOSIS.

The kidney is often involved. The lesion is apt to start in the pelvis and papiliæ. It results in cheesy areas like cysts, or foci that discharge into the pelvis of the ureter, causing pyonephrosis. The *symptoms:* Usually there is tuberculosis elsewhere in the genito-urinary tract, or in the lungs. The urine contains pus, albumin, tubercle bacilli, sometimes blood or cheesy matter. There are a large, tender kidney, attacks of pain like mild renal colic, and the general symptoms of a chronic tuberculosis.

Other genito-urinary tuberculosis is common, but the cases are more purely surgical.

QUESTION.

What are the lesions and symptoms of renal tuberculosis?

THE TREATMENT OF TUBERCULOSIS.

Preventive Treatment.—Discharges from tuberculous foci and everything contaminated by them should be carefully sterilized, preferably by burning. The sputum is the most common danger. It should not dry on handkerchiefs, but should be received in thick paper spit-cups or envelopes, which should be burned; or else into a china spit-cup containing carbolic acid (5 per cent.). Moist cloths should be used for "dusting" the room. After a patient leaves a house it should be disinfected. Dairy cattle should be tested periodically with tuberculin, and no tuberculous animal should be kept in a herd. Infected meat should not be sold. The patient should realize that his hands and lips are apt to harbor the bacilli, and he should clean them with special care and prevent needless contact with them.

In those predisposed to tuberculosis an out-of-door life is necessary. Catarrh or acute illnesses should be treated until the cure is complete, and cod-liver oil or iron should be used to restore vigorous health. All of these measures are easier with systematic notification of tuberculosis.

Treatment of the Patient.—*Open-air treatment:* The patient should be out of doors practically all the time, night and day, sleeping in a tent or on a balcony; or if this is not possible, in a room with widely opened windows. This treatment is effective almost anywhere.

Climate.—*Mountain climates* are good for cases that are not too weak, that have not very advanced lesions, emphysema, heart disease, a tendency to hemoptysis, or a marked nervous temperament. *Low, warm places* are best for the very nervous, the very weak, and others who should not go to the mountains. *Moderately elevated climates* are suitable for most cases. Much sunshine is always desirable.

Rest in bed, preferably in the open air, is advisable for those whose temperature stays above 101° F. Exercise without fatigue helps all who are strong enough. An abundance of nutritious digestible food is imperative, including milk and cream, meat, fats, and eggs. Forced feeding, even with the stomach-tube, may be resorted to. Overfeeding usually consists in giving much milk and eggs between meals and with meals. The appetite should be nursed carefully and stimulated with strychnin and tonics. Any treatment that lessens the appetite or digestion is harmful.

Drugs.—Creasote acts favorably by lessening fermentation in the stomach and bowels, by lessening the cough and sputum, and mitigating the lung inflammation. It is also an antipyretic. It may do harm by interfering with the appetite and digestion. Creasote carbonate, guaiacol or its carbonate, are less apt to disturb. Creasote may be administered by inhalation in a Robinson inhaler, worn almost constantly; or by the mouth, beginning with one or two drops after meals and increasing gradually to toleration, perhaps 20 to 40 drops a day, and then maintained at a little smaller dosage for many months. It may be given in various media, such as malt extract, in emulsion with sugar or with other drugs. Cod-liver oil is excellent, ℥ss–j, after meals. Other fats, especially cream, are good. Arsenic helps anemia, often in combination with iron; strychnin is a good tonic and stimulant. The various symptoms need special treatment. Tuberculin and antiserums are as yet used but little.

QUESTIONS.

Describe the preventive treatment of tuberculosis.
Discuss the open-air treatment, climate, feeding, and creasote.
Give the other treatment.

LEPROSY.

Etiology.—The specific cause is Bacillus lepræ, which is found in the lesions and the discharges from them. It is not known outside the body. Leprosy is transmitted by direct contact and by fomites. Infection is probably by inoculation or through the nasal mucous membrane. Leprosy is endemic in many places. It usually appears in young adults.

Lesions.—There are masses of round cells, epithelioid cells, and giant cells arranged in nodules or rather diffusely in the skin, nerve-trunks, mucous membranes, and sometimes in the viscera. The bacilli are in the cells and outside them. This tissue may ulcerate or cicatrize; the fingers and toes may slough. The *incubation period* may last for years.

Symptoms.—(1) *Tubercular leprosy:* At first certain areas in the skin are erythematous, hyperesthetic, and pigmented; later, white and anesthetic. They appear first on the extremities and face; later elsewhere. Tubercles appear in these areas; they are brown and anesthetic; may persist for years with great disfiguration, especially of the face; may ulcerate with loss of fingers and toes; or may be absorbed. They may occur in the nose, causing ozena; or in the larynx, causing hoarseness and aphonia, and may lead to inhalation pneumonia and death. (2) *Anesthetic leprosy:* Certain nerve-trunks, especially the ulnar, are nodular and tender. Along the distribution of these nerves there are patches of hyperesthesia and numbness; later of anesthesia. Trophic changes are present, and may result in bullæ, which may be followed by rather deep ulceration with contractures and loss of the fingers. The muscles waste. The tubercular and anesthetic types may be combined.

Prognosis.—The course is slow, often ten years in the tubercular type, and twenty or thirty years in the anesthetic type.

Treatment.—Segregation is probably advisable.

QUESTION.

Give the etiology, lesions, and symptoms of leprosy.

GLANDERS AND FARCY.

Etiology.—The specific cause is Bacillus mallei, which is present in the lesions and the discharges from them, in the urine and in the blood. The disease is transmitted by inoculation with the discharges from other cases. It is chiefly a disease of horses, and exceptionally is found in men who work around horses.

Lesions.—There are nodules of lymphoid cells and epithelioid cells and the bacilli. These are surrounded by a zone of exudative inflammation. They suppurate within two or three days, and if near the surface form an ulcer that becomes serpiginous. In deeper tissues abscesses result. The discharge becomes fetid. The lymph-nodes are inflamed and tend to necrose. There are also skin rashes. *Farcy* is a typical pyemia. The lesions appear first at the site of inoculation, and later in any tissue, but usually the intermuscular planes and lymph-vessels. The lumps in the lymph-vessels are called "farcy buds." In *glanders* the lesion is in the mucous membrane of the nose at the site of infection and the patient may die before pyemia develops.

Symptoms.—The *incubation period* is usually three to five days. In *acute cases* there is an abrupt onset of a severe fever, a leukocytosis, and the *rash*. This at first is papular, then pustular, and then forms crusts; it appears first on the face and spreads to the rest of the body; there are several crops; there may be much exudative inflammation around the pustule. There may be larger swollen areas with vesicles and blebs. Besides this there is in *acute glanders* a severe inflammation of the nose with a profuse discharge on the first day; on the second or third day ulcers appear, and the mucous membrane of the mouth and throat also is swollen; nose-bleed is common; the related lymph-nodes are swollen. In *acute farcy*, at the site of inoculation an abscess forms and discharges within two days. There are lymphangitis, farcy-buds, and inflamed lymph-nodes. Similar abscesses develop elsewhere, and the joints are swollen and painful. In *chronic glanders* the nose symptoms are less acute, the discharge often fetid and the process may involve the larynx, or later become generalized. In *chronic farcy* there is a series of abscesses, but less intense inflammation and less constitutional disturbance. The chronic forms may last several weeks or months, and end in a slow recovery or as acute glanders,

Prognosis.—Acute cases are fatal; chronic glanders is usually fatal. With chronic farcy some die of pyemia or acute glanders, others get well slowly after weeks or months.

Diagnosis.—In a doubtful case in man some of the discharge is injected into a guinea-pig, and Bacillus mallei may be recovered from its lesion.

Minute injections of mallein[1] seem to have helped some chronic cases.

QUESTIONS.

Give the etiology of glanders and farcy.
Describe the lesions.
Give the symptoms of the acute cases and of the chronic cases?
What is the prognosis?
What is mallein, and how is it used?

WEIL'S DISEASE.

This occurs in groups of cases, mostly in butchers and in summer. The **symptoms** are: Fever with abrupt onset; severe muscle pains; jaundice, which may be slight or intense; the liver is somewhat enlarged and tender; the spleen is enlarged; there are albumin and sometimes blood in the urine; the fever is remittent, lasts about two weeks, and falls by lysis. The **prognosis** is usually good, but there may be delirium, coma, convulsions, and death.

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[1] Mallein is the filtrate through porcelain, of an old broth culture of the bacillus of glanders, that has been killed by heat. In lower animals it is used for diagnosis.

DISEASES DUE TO ANIMAL PARASITES.

WORMS.

I. CESTODES OR TAPEWORMS.

(A) Intestinal Cestodes.

(1) **Tænia mediocanellata.**—The adult is 15 to 20 feet long; the head has 4 suckers but no hooks; the mature segments are about ¾ inch long; the uterus has many dichotomous branches; the ova are about 30 μ long. Man is the definitive host. The alternate host is cattle, and in them the cysticerci are found in the muscles, especially of the jaw, where they retain their vitality for years.

(2) **Tænia solium.**—The adult is 6 to 12 feet long; the head has hooks and 4 suckers; the mature segments are about ½ inch long; the uterus has fewer lateral branches; the ovum is much like that of the other form. The alternate host of this worm is rarely man himself; usually it is the pig, the embryos lodging in the muscles (measly pork).

(3) **Bothriocephalus latus** is a very long worm (30 feet). It is very rare in America, and is gotten from fish.

Symptoms.—The segments of the worms are passed with the stool or crawl through the anus at other times. The ova are found in the stools. There may be no other symptoms; but often there are attacks of nausea, vomiting, diarrhea, and colic There may be fever, anemia, or nervous symptoms.

The *diagnosis* is made by finding the segments or the ova in the stools.

Treatment.—Preventive treatment: Inspection of meat at abattoirs; thorough cooking of meat. All tapeworms from patients should be killed. *Curative treatment:* The patient should fast for a day and take a mild laxative. Then he should take aspidium, pelletierin tannate, cusso, or other anthelmintic; and half an hour later a very brisk purge. If the head is not expelled, the worm grows again, and after two to six months reaches maturity. It should then be attacked again.

(B) Visceral Cestodes.

(1) Occasionally man serves as the alternate host of Tænia solium. The larva is called **cysticercus cellulosæ.** The ova enters the stomach with water or food, or possibly from an intestinal worm by retroperistalsis in vomiting. The shell is digested off. The embryo penetrates the stomach-wall and lodges in various places, where it develops into cysticerci (bladder worms). These may die and be encapsulated or calcified. During the preliminary stages there may be digestive disturbances. When the embryos lodge in the tissues there may be symptoms due to the mechanical presence of foreign bodies. They may be found in the subcutaneous and intramuscular connective tissue, in the membranes of the brain or cord, in the eye or elsewhere. There is no treatment except excision when this is possible.

(2) **Echinococcus Disease.**—This is rare in the United States. Tænia echinococcus is a tapeworm of the dog. The larvæ live in pigs and cattle and may infect man. The embryos are ingested, the shell is digested off, the freed embryo penetrates the stomach-wall, lodges somewhere and develops. At one pole there are six hooks, which are cast off. The embryo then develops into a cyst with two layers, the outer a dense capsule, the inner the granular or parenchymatous layer. The cyst contains a clear liquid, specific gravity 1005 to 1009, without albumin. The host reacts with a productive inflammation, and ultimately a fibrous capsule may be formed. The cyst grows larger, the inner layer develops twelve or more buds, each of which becomes a daughter cyst which is like the parent cyst and floats freely inside of the parent cyst. These daughter cysts form another lot of cysts within themselves (granddaughter cysts). From the inner layer of all cysts buds appear which develop into scolices. A scolex consists of a head and neck like those of the adult worm, and in the intestine of a dog it may grow to maturity. One embryo may form many thousands of scolices. The scolices may live latent in the tissues for years, or the cysts may dry with death of the scolices and the formation

of a granular mass that may calcify. The cyst may rupture and discharge scolices into some cavity, vessel, duct, or externally. It may suppurate. Some echinococcus cysts become very large. In man the usual site is the liver, less often the lung, urinary organs, or brain.

In the *liver* it forms a tumor which is elastic, may fluctuate or give hydatid fremitus. The aspirated liquid may contain hooks but no albumin. The health of the patient is little affected. The cyst may rupture or suppurate. In the *lung* hydatids are apt to cause pneumonia or gangrene. Little cysts may be spit out. There may be hemoptysis or the cyst may rupture. Cysts of the *pleura* may give the signs of an irregular, sacculated pleurisy with effusion. The aspirated liquid may contain hooks but no albumin. The general health may be good. In the *kidney* it resembles hydronephrosis. In the *brain* it resembles a tumor.

Treatment.—Surgical treatment may be followed by recovery.

QUESTIONS.

Describe the beef tapeworm and the pork tapeworm.

Give the symptoms, diagnosis, and treatment in man.

Give the source and nature of cysticerci and the symptoms caused by them in man.

Give the source and natural history of echinococcus and the symptoms caused in man.

Describe the cyst contents and the diagnostic value of its analysis.

II. TREMATODES.

(1) **Distomum hepaticum,** the liver fluke, may cause an enlarged liver, jaundice, ascites, diarrhea and emaciation. The ova occur in the stools.

(2) **Distomum pulmonale,** the bronchial fluke, is the cause of endemic hemoptysis in Mongolian countries.

(3) **Dystomum hæmatobium** (Bilharzia hæmatobia), the blood fluke, is very common in man in Egypt. The adult worms cause no symptoms. The ova cause a peculiar hematuria or dysentery, and are found in the urine or stools.

III. NEMATODES.

Nematodes have no intermediate host. The eggs are ingested with water or uncooked food, or are carried to the mouth by fingers.

(1) **Ascaris lumbricoides** is the "round worm." The male is 5 to 6 inches long, the female 8 to 10 inches long. They look somewhat like an earthworm, live in the small intestine, and may crawl out of the anus, the mouth, the nose, or into the bile-ducts. Usually there are only one or two, but there may be many. The ova are found in the stools. *Symptoms* may be insignificant or there may be nervous symptoms, such as restlessness, picking at the nose, pruritus ani, grinding the teeth, or muscular twitching. A worm may lodge in the bile-duct, causing jaundice, or in the larynx, causing asphyxia. The *diagnosis* is made by finding the eggs or the worm. *Treatment:* Santonin gr. ss–ij for an adult, nightly for three or four nights, combined with a purge, is very effective.

(2) **Oxyuris vermicularis,** the "thread or seat worm," is a fine, white worm, $\frac{1}{5}$ (male) to $\frac{2}{5}$ (female) inch long. It lives in great numbers in the colon and rectum; the ova are found in the stools; the worms may crawl out of the anus at night. They cause pruritis ani, restlessness, sleeplessness, perhaps anemia. *Treatment:* Large high enemata of brine, quassia water, or vinegar, daily for several days; with santonin and purges to reach those higher up. An ointment of mercury at the anus prevents itching and migration of the worms.

(3) **Uncinariasis** is due to the **Ankylostoma duodenale** (Uncinaria duodenalis), a small worm that lives in the upper part of the small intestine. It is very common in Egypt, Porto Rico, India, and Brazil, and occurs in the southern part of the United States. The *symptoms* are anorexia, diarrhea, tympanites, colic, with ova in the stools; a progressive and severe anemia; eosinophilia; great muscular and mental sluggishness. The asthenia and anemia may be fatal. The *diagnosis* is made by searching for the ova in the stools. This is an important disease. *Treatment:* A light diet and mild laxative for a day or two; then thymol ʒj in the morning, followed after an hour by a *saline* purge. Alcohol and oils must be avoided. The anemia should be treated.

(4) **Trichiniasis.** Trichina spiralis may be parasitic in man. It is derived from eating imperfectly cooked, infected pork; the pig is supposed to get it from eating infected rats. The embryos are eaten with the pork, the capsule is digested off in the stomach; they develop in the small intestine in

about two days. The adult female is ⅛ inch long, the male shorter. They pair, and in about a week the female begins to give birth to embryos, and in a few weeks produces a thousand or more; then the adult dies. The embryos promptly enter the intestinal lymph-spaces, and finally reach the voluntary muscles, where they lodge within the sarcolemma and develop. The host reacts with an interstitial myositis, forming a fibrous-walled cyst which encloses one or two embryos. The embryos may live several years or may die and become calcified. The cyst is a dot, just visible to the naked eye. *Symptoms:* During the *first few days* there are anorexia, diarrhea, and abdominal pain. In the *second stage* there is a myositis, with tenderness, stiffness, and swelling and impaired movement. This is more common in the muscles of mastication, of the larynx, the intercostals and the diaphragm. There are edema of the face and eyelids, a remittent or intermittent fever, a leukocytosis with marked eosinophilia, anemia, and more or less prostration. With *few parasites* there may be almost no symptoms. *Moderate cases* recover after three weeks. *Severer cases* last longer and may develop the typhoid state. *Convalescence* is slow. Some cases die of exhaustion, diarrhea, peritonitis or pneumonia from the fourth to the sixth week. The *diagnosis* is based on the sequence of digestive and muscular symptoms with edema of the face and eosinophilia. Adult worms possibly may be found in the stools. Embryos may be found in the suspected pork or in a piece of affected muscle excised for examination. This is an important disease. *Treatment: Preventive:* Cook all pork thoroughly; also all pork should be examined at the abattoirs. *Of the disease:* If seen early, thymol, santonin, and purges may help; later, merely symptomatic treatment.

(5) **Filariasis.**—Filaria Bancrofti and F. nocturna. The adult worm is called Filaria Bancrofti. The female is 3 inches long, the male shorter; they live in the lymphatics of the trunk and limbs. The embryos, called F. nocturna, are discharged into the lymph, get into the blood-vessels, and are found in the peripheral blood at night only. In the daytime they stay in the deeper organs. Such an embryo is cylindrical, about 300 μ long and 7.5 μ in diameter, and has a *very active* lashing motion. A certain mosquito ingests these

with the blood, they develop in its body, probably are discharged into water from the dead body of the mosquito, and are drunk with the water by man. Further details are not known. This is very common in China, India, Brazil, etc. The embryos cause no symptoms except their presence in the blood at night. The adult worms usually cause no symptoms, but sometimes they block the lymphatics, causing a varicose condition in these vessels, and leading to (1) endemic chyluria, or (2) lymph scrotum, or (3) elephantiasis arabum.

Treatment.—The enlarged lymph-nodes with the adult worm may be excised.

Other filariæ are described, but are less important.

QUESTIONS.

What is the mode of infection with round worms and thread worms? Give the symptoms, the diagnosis, and the treatment of each.

Give the cause, symptoms, diagnosis, and treatment of uncinariasis.

Give the natural history of Trinchina spiralis.

Give the symptoms of trichiniasis.

Give the prognosis, the preventive treatment, and the early treatment of the disease.

Give the causes, symptoms, and diagnosis of filariasis.

ARACHNIDS.

(1) **Scabies** (itch) is due to Acarus scabiei, which is just visible to the naked eye; the impregnated female burrows in the skin, deposits eggs, and dies at the end of the burrow. The larvæ also burrow. The burrow is a fine line, perhaps $\frac{1}{8}$ inch long. The usual site is in soft skin between the fingers, at the folds of the elbows, etc. Papules, vesicles, pustules, scratch-marks, and the result of consequent infection are present. There is much itching, especially at night. The disease is contagious. *Treatment:* Sulphur ointment is thoroughly rubbed in, after a thorough scrubbing in a hot bath. This is repeated on three or four successive nights.

(2) **Acarus folliculorum** is very common in comedones, but causes no symptoms.

INSECTS.

(1) **Pediculosis Capitis.**—Pediculus capitis, or head louse, is about $\frac{1}{10}$ inch long, lives on the scalp, and deposits ova on

the hairs (nits). It causes itching by crawling on the scalp and by puncturing it for blood. Scratching results in a serous crust; there may be eczema extending to the neck and behind the ears; the lymph-nodes may be enlarged and tender. The tincture of delphinium and ether in equal parts, or turpentine or kerosene, is rubbed on at night. In severe cases the hair should be cut.

(2) **Pediculosis Corporis.**—Pediculus corporis or vestimentorum is larger than the head louse, lives on the clothing, attacks the hairless parts of the body, especially where the clothes press at the waist and shoulders. It leads to itching and papules, scratching and more or less infection of the skin. Later there may be pigmentation. The *diagnosis* is made by finding the lice *in the clothing. Treatment:* Sterilize the clothing and bathe the patient.

(3) **Pediculosis Pubis.**—Pediculus pubis, or crab louse, lives on the pubic and other hairy regions, except the scalp, and deposits ova on the hairs. It causes itching, papules, and lesions from scratching. *Treatment:* Scrub the skin two or three times a day and apply mercurial ointment.

(4) The bite of **Cimex lectularius,** or bed-bug, forms a wheal with a dot in the middle, and causes any degree of itching.

(5) The bite of **Pulex irritans,** or flea, is a petechia surrounded by redness. There may be more diffuse erythema or urticaria.

(6) **Pulex penetrans,** the sand flea, or jigger, is very small and burrows in the skin, usually of the feet. A vesicle or pustule develops. The insect should be removed with a needle.

Myiasis is a condition in which only the larvæ of insects (maggots) are present. They may be found in wounds (Myiasis vulnerum); in the nose (M. narium); in the ear (M. aurium); on the vulva (M. vulvæ); or in the alimentary canal (M. interna), with maggots in the vomitus or stools. The *treatment* for the external kinds is to clean the wound or cavity; for the internal, purges.

QUESTIONS.

What are the diagnostic points and the treatment of the disturbances *due to* lice, scabies, etc. What is myiasis?

HEAT-STROKE AND THE INTOXICATIONS.

HEAT-STROKE.

(1) **Heat exhaustion** follows hard exertion in intense heat, and is probably due to vasomotor paralysis. *Symptoms:* There is rather abrupt prostration, with more or less collapse, restlessness, and perhaps delirium. The skin is cool, and the temperature subnormal; recovery is the rule. *Treatment:* Rest in a cool place, perhaps a hot bath, or stimulation and vasoconstrictors.

(2) **Thermic fever** (insolation, sunstroke) is seen after a few days of very hot, humid weather, following exertion, and especially in alcoholics. *Lesions:* Rigor mortis and putrefaction are early; the blood is liquid; venous congestion is intense; and there are degenerations in the liver, kidneys, brain, and cord. This is probably due to a disturbance of the heat center. *Symptoms:* After a variable period of vertigo, weakness, and headache, the patient suddenly becomes very ill. The skin is very hot and flushed, it may show petechiæ; there are vomiting, diarrhea, incontinence of feces and urine, a fast, full pulse, stertor, coma, or perhaps active delirium, contracted pupils, and relaxed muscles (sometimes general convulsions), and a temperature of 107°–112° F., or even higher. In favorable cases the temperature falls to normal in a few days and the symptoms clear. There may be such sequels as susceptibility to hot weather, restlessness, headache, weakness, imperfect memory, a lack of concentration, or peripheral neuritis. The fatal cases die in coma or convulsions with failing heart, edema of the lungs, and hyperpyrexia in a few hours or a day or two. The mortality is 30 to 50 per cent.

Asphyxial cases suddenly fall unconscious and die with intense venous congestion and heart failure in a few minutes. The temperature is elevated.

Treatment.—Baths in ice water and later in cold water if the temperature rises. Besides this, stimulation and symptomatic treatment are needed. Asphyxial cases need very prompt blood-letting and stimulation.

QUESTIONS.

Give the symptoms and treatment of heat exhaustion.

Give the etiology, symptoms, sequels, and treatment of thermic fever.

ALCOHOLISM.

(1) **Acute Alcoholism.**—The *symptoms* suggestive of coma due to alcohol are a cold, clammy skin, signs of previous vomiting, retention of urine, or perhaps incontinence of feces and urine, a soft pulse, either slow or fast, a subnormal temperature (96° F.), stertor, alcoholic breath, pupils that are equal and dilated (at least after slapping the temples), coma (but pressure on the supraorbital notch or shouting in the ear arouses the patient to incoördinate utterance) flabby muscles, and sometimes signs of chronic alcoholism or the history of much drinking. After a few hours the condition passes off, leaving headache, muscular weakness, anorexia, and often vomiting. Some cases are fatal, with deeper coma and failing heart. *Treatment:* Apomorphin hydrochlorate, gr. $\frac{1}{10}$ hypodermically; lavage with hot water; hot enemata; stimulation with ammonia, digitalis, etc.

(2) **Acute alcoholic mania** occurs after drunkenness in neurotic persons. It is violent, often homicidal, and passes off in a few hours or a few days.

(3) **Chronic Alcoholism.**—The toper is sallow, tremulous, has acne rosacea, a coated tongue, foul breath, nausea and vomiting, especially in the morning, mental dulness, a lack of will power, muscular fatigue and flabbiness. There may be symptoms of other conditions due to alcohol, such as chronic gastritis, cirrhosis of the liver, arteriosclerosis, pachymeningitis, alcoholic dementia, maniacal outbreaks, delirium tremens, multiple neuritis or chronic nephritis. There is also a predisposition to many acute infections.

(4) **Delirium tremens** is seen in chronic cases after a debauch with little food; or after sudden abstinence for a

few days, in acute fevers, and after shock following an injury or operation. The patient becomes restless and fearful, develops hallucinations of sight and hearing (rats, snakes, or other animals, and threatening sounds), great terror, a very active delirium with loud talking and shouting, sleeplessness, marked tremors, a rapid, feeble pulse, and a temperature from 101° to 103° F. The symptoms often abate after two or three days with the onset of sleep, and there is a gradual return of appetite, sanity, and strength. Fatal cases may develop the typhoid state in a few days, and die of exhaustion or of complicating pneumonia, heart failure, or possibly cerebral hemorrhage. *Treatment:* Most authorities give no alcohol to uncomplicated cases. Chloral, gr. x–xx, bromids, gr. xxx with digitalis (♏ x–xv of the tincture), every four hours, may be given, or trional gr. xxx, or hyoscin. The patient should be fed with liquids by mouth or rectum. With pneumonia alcohol in moderate amounts is usually given. The lungs should always be carefully examined.

(5) **Dipsomania** is a condition in which there are attacks of uncontrollable thirst for drink, lasting a few days or weeks. These recur at varying intervals. During the interval there may be no desire for alcohol.

QUESTIONS.

What are the symptoms and treatment of alcoholic coma?

What are the symptoms, prognosis, and treatment of chronic alcoholism and delirium tremens?

What is dipsomania?

CHRONIC OPIUM-POISONING (Morphinism).

The **symptoms** are pallor; a sallow, dry, wrinkled, itching skin; anorexia, constipation, or attacks of diarrhea, colic; heart pain; pupils dilated except when contracted by the drug; insomnia, mental depression, irritability, mendacity, a blunted moral sense, restlessness, tremor; progressive anemia, and loss of flesh and strength. At times there are attacks of chills, fever, and sweating, sometimes with delirium and albuminuria. Finally death ensues from asthenia or an intercurrent disease. Many persons take opium habitually without ill health, but usually the dose is steadily increased.

The **diagnosis** is sometimes helped by the presence of scars from the needle, but the patient's word is utterly unreliable. The *sudden stoppage* of the drug sometimes causes nausea, vomiting, diarrhea, abdominal pain, a weak heart action, extreme restlessness, and sometimes collapse.

Treatment should be carried out in a sanitarium. The mind and the muscles must be preoccupied. Good food is necessary; cold baths are indicated. The opium is tapered off and stopped after ten days. For the restlessness use bromids, cannabis indica, sulphonal, etc. For the heart strychnin, physostigma, ammonia, and digitalis. Treatment must be continued for many weeks, and then relapses are usual.

QUESTION.

Give the symptoms and general principles of the treatment of chronic opium-poisoning.

PLUMBISM.

This follows the continued absorption of small amounts of lead (*a*) from water, beer, or wine that contains dissolved lead; (*b*) in painters, plumbers, glaziers, or workers with metallic lead (from dirty hands). It may appear after a few weeks or many years of exposure, and is usually in adult males.

Lesions.—The muscles are yellow, fatty, and fibrous; the nerves are degenerated; the cord is rarely affected: there may be enterocolitis.

Symptoms.—There are pallor, a dotted blue line in the edge of the gum (this is diagnostic); anorexia, constipation, protracted attacks of abdominal colic, with a slow pulse and high tension (*lead colic*). There may be *paralysis*, usually of both musculospiral nerves with wrist-drop; or of the peroneal muscles with foot-drop; or less often of the shoulder or other muscles. The muscles atrophy and may give the reaction of degeneration, but sensation persists. *Encephalopathies:* There may be optic neuritis or neuroretinitis with impaired sight; hysteria, convulsions, epilepsy, trance-like attacks, attacks of delirium or hallucinations, or tremors. Symptoms from one or more of these groups may be present. They come on slowly or acutely. There is a *tendency* to arteriosclerosis, *gout,* and interstitial nephritis.

The **prognosis** usually is good.

Treatment.—*Preventive:* Avoid the causes by care with liquids in lead-containers and in dangerous trades by washing the hands carefully before meals. *Curative:* Except in very acute cases, potassium iodid, gr. xv–xxx, is given daily. For constipation, salts. For colic, hot fomentations or morphin if necessary. For the paralyses, massage, galvanism, or strychnin injection.

QUESTIONS.

Discuss the etiology of lead-poisoning.

What are the symptoms?

How is the diagnosis made from other forms of colic or paralysis, or other nervous disturbances?

To what other diseases does lead especially predispose?

What is the treatment?

CHRONIC MERCURY-POISONING.

This is found in workers in mercury mines or manufactures, and in those who take mercury as a drug. The susceptibility varies greatly.

The **symptoms** are: (1) *Stomatitis,* which is marked by salivation, and tenderness of the salivary glands, swollen, bleeding gums, loose teeth, a swollen tongue, and fetid breath. In extreme cases there may be necrosis of the jaw. There are anorexia, vomiting, and diarrhea. (2) *Nervous symptoms:* There is a characteristic *intention tremor* appearing first in the lips and tongue, but spreading to other parts of the body; at first it is fine, later coarse. There may be *paralyses* of the limbs from neuritis. Some cases have irritability, headache, loss of mental power, delirium, even coma or convulsions. With proper treatment most cases recover, but sometimes there is permanent mental damage.

Treatment.—Mercury absorption must be stopped. Potassium iodid and sulphur baths are used. For the stomatitis, astringent and cleansing washes.

QUESTION.

What are the symptoms of mercurialism?

CHRONIC ARSENIC-POISONING.

This is found in (*a*) arsenic eaters; or (*b*) in workers in certain trades, mines and factories, or glass-works; (*c*) the arsenic may come from dyes, or (*d*) it may be taken medicinally. Susceptibility varies and toleration may be developed.

The principal **symptoms** are puffy eyelids; vomiting and diarrhea; a pale or pigmented skin, or skin eruptions, such as herpes; peripheral neuritis with numbness, later pain and paralyses (foot-drop and later wrist-drop); a fine tremor; there may be headache, melancholia, or hysteria. Most cases recover.

The **treatment** is to stop the arsenic absorption, to give potassium iodid and purges, to use the customary treatment for the neuritis.

QUESTIONS.

What are the sources of the arsenic in chronic poisoning?
Give the symptoms and treatment.

FOOD-POISONING.

Animal Food.—*Milk* or *meat* may contain unidentified organisms that cause poisoning after an incubation period of several hours or a day or two. At other times they may contain ptomains that poison within a few hours. All putrefied meat is not toxic—*e. g.*, high game. Milk not kept at a low temperature, or cheese, or ice-cream (particularly after melting and re-freezing) may produce within a few hours vomiting, purging, abdominal colic, vertigo, headache, chilliness, and marked prostration. The worst cases have a subnormal temperature, collapse, and death. Meat, usually sausage, may cause similar symptoms. The *diagnosis* is aided by the simultaneous appearance of these symptoms in several persons who had eaten together.

Fish and *mussels* may cause: (1) vomiting, diarrhea, colic, and tenesmus; or (2) nervous symptoms, incoördination, convulsions, paralyses, perhaps dilated pupils, collapse and death in a few hours. In either type there may be urticaria, itching, dyspnea, or delirium. *Treatment.*—Careful prophylaxis is important. In the attack the alimentary canal should be emptied by lavage, purging, and colon irrigation. Stimulation often is needed.

Vegetable Poisoning.—Ergotism, lathyrism, and pellagra, due to ergot (a parasite on rye), to fermented chick-pea and fermented maize, respectively, are hardly found in America. Certain degenerations in the nervous system with their symptoms are present.

QUESTION.

Give the symptoms of poisoning by animal foods. Give the prognosis and treatment.

CONSTITUTIONAL DISEASES.

CHRONIC RHEUMATISM.

Etiology.—Usually occurs after middle life, associated with habitual exposure. *Lesions:* At times the joints may show slight effusion. The capsule and ligaments may become thickened with fibrous tissue, which causes creaking and some limitation of movement. The joint-lesion often is slight. Muscular wasting and perhaps chronic endocarditis may occur.

Symptoms.—The onset is slow. The joint is painful and stiff, less so after exercise, more so in damp weather. Swelling and redness are slight, but there may be persistent aching in the joints. It is usually polyarticular and the general health is little disturbed. There are exacerbations and remissions, sometimes restricted movement of the joints and chronic endocarditis. Recovery is rare, but life is seldom shortened.

Treatment.—A dry, warm climate during the winter; generous feeding and careful hygiene; courses of baths at hot springs are helpful. The local treatment consists of dry heat (300° F.), counterirritation with the cautery or blisters, massage and passive movement. Drugs are of little use.

QUESTION.

Give the etiology, lesions, symptoms, and treatment of chronic rheumatism.

ARTHRITIS DEFORMANS.

Etiology.—This disease is neither rheumatism nor gout. It is more common from twenty to forty years, in females, after grief or worry, and in the poor. It is supposed to be either (1) a trophoneurosis or (2) a chronic microbic infection.

Lesions.—Early in the disease some joint-effusion may be present. Later, the cartilages are softened and eroded at the middle of the joint, while at the edges of the joint they contain nodules which may be calcified or ossified Where the cartilage is eroded the bone becomes smooth and dense (eburnated). Near the edges of the joint bony outgrowths (osteophytes) develop. The synovial fringes and the ligaments are thickened with fibrous tissue and nodules that may become cartilaginous, calcified, or ossified. There are more or less immobility and deformity of the joint, and sometimes subluxation. The muscles atrophy much, and the nerves around the joint may degenerate. The heart is not affected.

Symptoms.—(1) The *acute polyarticular* form is rare. It occurs usually in women from twenty to thirty years old, with or after pregnancy, but it may follow the menopause. Many joints are swollen, red, painful, and tender. There is usually effusion. There may be slight fever. The attacks may last several weeks, with anemia and mental depression. It may recur with the next pregnancy or become stationary or chronic. (2) The *chronic polyarticular* form is important, and involves many joints of the hands, spine, jaw, etc. There are joint-pains, tenderness, impaired mobility, grating, and the nodules. The characteristic hand has the terminal phalanges partly flexed, the fingers bent to the ulnar side, and evident nodules, especially at the distal ends of the second phalanges. The thumb is seldom involved. The toes may be similarly deflected toward the fibular side. There may be marked locking of the joints of the spine or of the jaw. There are paresthesias, a shiny skin about the joints, and marked muscular wasting, with at times slight fever. The general health is quite good. The course may be arrested at any point, or may be steadily progressive, or marked by exacerbations and remissions. Finally the patient may be practically motionless. (3) *Localized forms:* (*a*) The *monarticular* form is more common in old men, in the hip (morbus coxæ senilis), or shoulder, or knee. Subluxations are common. It may involve the whole spine or a part of it, with rigidity. (*b*) *Heberden's nodes* are more common in middle-aged women, near the distal joints of the fingers. At times they may be tender and painful, with redness and swelling of the skin. This form seldom spreads and may cease to trouble the patient except by the impaired movement. (4) In chil-

dren, especially girls, before the second dentition, it may attack the knees, wrists, or cervical spine, beginning rather acutely in one or two joints and spreading to others. The joint-symptoms are rather typical; but there are also general enlargement of the lymph-nodes and spleen, and often anemia and deficient development.

The **treatment** is like that of chronic rheumatism, but is rather ineffective.

MYALGIA (Muscular Rheumatism).

Etiology.—This is more common in the rheumatic or gouty; in adults; in males. It may be induced by exposure to cold, especially after exercise. Repeated attacks are common.

Symptoms.—There is rarely slight fever for two or three days. In the muscles affected there is a sudden onset of pain, which may be severe, and is induced by movement of the muscle, and of tenderness and rigidity. It lasts a few hours or days, or may persist with remissions for months. Many special names have been given to the various forms. *Lumbago* involves the lumbar muscles; *Pleurodynia*, the intercostals with painful breathing. It is usually on the left side, is more extensive than neuralgia, lacks the tender points, and there are no râles. It may involve the pectorals and the serratus also. Some cases of *torticollis* are myalgic. *Cephalodynia* is rare. It involves the muscles of the scalp. *Omodynia* involves the shoulder muscles. There are also *scapulodynia* and *dorsodynia*. *Abdominal rheumatism* involves the abdominal muscles; *rheumatic myositis*, the muscles of the limbs.

The **prognosis** is good, but the pain may be protracted, and subsequent attacks are common.

Treatment.—*Drugs:* Salicylates; the injection of strychnin; or perhaps morphin. Potassium iodid or tonics may help the chronic cases. The local treatment is the most important—wet or dry heat, the Turkish bath, the thermocautery, galvanism, blisters, acupuncture, and massage. An even temperature of the skin should be maintained by woollen clothing.

QUESTION.

Give the etiology, symptoms, and treatment of myalgia.

GOUT.

Etiology.—Usually appears in middle life; rather more commonly in males; with alcohol, especially malt liquors and sweet wine; with overeating and deficient exercise; chronic lead-poisoning; and in certain countries. Heredity strongly predisposes, and the symptoms appear earlier in hereditary cases. An acute attack may be induced by overeating, overdrinking, an emotion, injury, or have no discoverable cause.

There is excessive production or defective elimination of *uric acid*, or both. Uric acid occurs in the blood in gout as a sodium quadriurate, which is quite soluble; it is the ordinary acid urate of the urine. Under certain conditions this is changed to sodium biurate, a very insoluble form, which is the type found in the joints, in tophi, etc. It is a mechanical irritant, but probably not a chemical poison. There are many theories as to the rest of the pathology of gout.

Lesions.—There is an excess of sodium urate in the blood, with crystalline deposits in the cartilages of involved joints, some necrosis of cartilage, and at times an effusion into and around the joints. In early attacks, all except the lesion in the cartilage resolves perfectly. Later there may be exostoses around the joint and deposits of biurate of sodium in the ligaments, tendons, bursæ, ear, and the subcutaneous tissue of the fingers and toes (tophi or chalk stones). The soft structures of the joint are thickened and there are deformity and limited movement. It usually begins in the metatarsophalangeal joint of the great toe, and later attacks the other joints of the extremities. There may be ulceration or suppuration at the tophi. Associated with these changes there is a strong tendency to chronic interstitial nephritis with deposits of biurates in the kidney, to arteriosclerosis and other chronic productive inflammation.

Symptoms.—**Acute Gout.**—*Prodroma* are usually present. They are irregular pain in the joints, indigestion, irritability, perhaps sore throat, or asthma. The *onset* of the attack is abrupt, usually in the early morning, the patient being awakened by intense inflammation in his great toe. The toe is red, extremely painful, very tender, much swollen, and hot. There is fever (102°–103° F.). The urine is diminished, heavy, may contain some albumin and perhaps sugar. The mind is clear, but irritable. After two or three hours all the symptoms remit, to return nightly with diminishing intensity for about a week. There may be desquamation over the joint. After the attack the general health may be unusually good. Other attacks occur at intervals. The gout is apt to become chronic with incomplete remissions. Before and during the first part of an attack the urates and phosphates in the urine are diminished. During the latter part of the attack they may be normal.

Retrocedent Gout (*Suppressed Gout*).—Rarely the joint-symptoms may abate and be replaced by some of the following symptoms: Abdominal pain, vomiting, diarrhea; faintness; heart pain and irregularity, palpitation, dyspnea, or acute pericarditis; active delirium, coma, or apoplexy. The patient becomes very ill and may die.

Chronic Gout.—After a variable number of acute attacks which become less typical and more protracted, there are longer or shorter exacerbations, very like subacute or chronic rheumatism, which sometimes are induced by diet or emotions. The joints show the lesions mentioned above, and there are nephritis, arteriosclerosis, etc. The patient may die of uremia, pneumonia, inflammation of the serous membranes or apoplexy.

The *diagnosis* is made by the history of the predisposing factors and of previous typical attacks, and by the presence of tophi.

Irregular or **abarticular gout** includes various symptoms that occur without gout in many persons, but seem more common in those with a gouty heredity or tendency. They include eczema, psoriasis, pruritus, and other skin affections; attacks of headache with indigestion and constipation, or with diarrhea and colic; flatulence; persistent high tension of the pulse with its sequels; emphysema, chronic bronchitis; headache, migraine, neuralgias, apoplexy, perhaps meningitis; iritis, glaucoma. The urine may be very acid and high colored with uric acid crystals on standing (this excess may be periodic, and is then called a "uric-acid shower"). There may be intermittent or constant glycosuria, oxaluria, gravel and stone, chronic interstitial nephritis; or cystitis, urethritis, or endometritis; or irregular pains in the bones, joints, and muscles.

Prognosis.—The acute attack passes, but gives promise of others. Retrocedent gout may kill. After years of chronic or irregular gout the patient is apt to die of some sequel.

Treatment.—*The acute attack:* The foot is elevated and wrapped in cotton. *Drugs:* Colchicum wine, ♏xx–xxx every four hours until the pain goes or intestinal symptoms appear. Sodium salicylate, ʒj or more a day; alkalies. Morphin may be necessary at first. The diet is liquid with much water. *After the attack* the patient should drink much water and no alcohol; should eat moderately, with little red meat, and should take systematic exercise. Treatment at baths is excellent. Bitter tonics, potassium iodid, or piperazin, gr. x, three times a day may help.

QUESTIONS.

Give the etiology, the lesions, and the symptoms of acute and chronic gout.

Discuss uric acid and gout.

What is irregular gout?

Give the prognosis of gout.

Give the treatment of gout.

DIABETES MELLITUS.

Etiology.—This is a disordered metabolism of carbohydrates variously ascribed to disturbance of the pancreas, the central nervous system, the liver, or the intestinal villi. It is more common in males, in adult life, with overeating, sedentary habits, obesity, and with inheritance of diabetes itself or of gouty tendencies, and in Jews. It may follow injury to the head or elsewhere, worry, fatigue, exposure, perhaps infectious diseases, or arthritis deformans.

Lesions.—The *pancreas* often is diseased. It may show chronic interstitial pancreatitis involving the islands of Langerhans, atrophy, fat necrosis, or tumors. In the *nervous system* there may be hemorrhage, tumors, or sclerosis involving the floor of the fourth ventricle. These lesions are supposed to be causative. The blood contains more than the normal 0.15 per cent. of glucose. Other lesions are mentioned with the symptoms.

Symptoms.—Almost any of the symptoms may be noticed first. The *onset* is usually insidious, but may be rather abrupt. There are a dry, harsh skin, a red, dry, glazed tongue, caries of the teeth, gingivitis; excessive appetite and thirst, constipation with attacks of diarrhea; a temperature often subnormal; restlessness and mental depression; and progressive emaciation and weakness. The urine is increased to from 6 to 30 pints a day or even more, is colorless, acid, of a sweet odor, specific gravity 1020 to 1040 or higher, may contain a trace of albumin; it contains sugar (glucose), $\frac{1}{2}$ to 5 per cent. or more; the daily output of glucose varies from an ounce to a pound or two. The urea and phosphates are much increased. At times there may be acetone, diacetic acid, or less often β-oxybutyric acid in the urine.

Complications and less common symptoms: In the skin, boils; carbuncles; eczema; pruritus, especially of the external genitals; gangrene, especially of the feet; or perforating ulcer. Gastritis. Pneumonia, or gangrene of the lungs (without fetor), often tuberculosis; sometimes fat-embolism. *Diabetic coma:* (1) After exertion there may be weakness, stupor, coma, and death in a few hours. (2) After indigestion there may be dyspnea (air hunger), then headache, delirium, coma, a rapid feeble pulse, a fruity breath, and death in one to five days. (3) There may be an abrupt onset of headache and vertigo, then coma, and death in a few hours. Any of these forms may possibly follow a deficiency of carbohydrates in the food. There is an excess of β-oxybutyric and diacetic acids in the urine, and sometimes a decrease of the sugar. Coma in diabetes may result from uremia or apoplexy. *Diabetic tabes* is a neuritis with cramps in the calves, no knee-jerks, a "steppage" gait, paralysis of the foot extensors, and perhaps optic nerve atrophy. In the *eye* there may be cataract, retinitis, optic atrophy, etc. In the genito-urinary system there may be interstitial nephritis, rarely pneumaturia from fermentation in the bladder, or impotence, sterility, abortions, vulvitis, or balanitis.

Course, Clinical Varieties. and Prognosis.—*Lipogenic* or *dietetic glycosuria* is hardly real diabetes; it is usually transient, in the obese, and is perfectly controlled by diet. *Pancreatic diabetes* is rapid and severe, and is marked by defective assimilation of fats and proteids. *Phosphatic diabetes* is a poorly understood condition, with the general symptoms of diabetes and a great excess of phosphates in the urine, but glycosuria may be intermittent. The *mildest cases* of diabetes may be well controlled by diet, and after many months an ordinary diet may safely be resumed. The *ordinary case* of diabetes is not cured, but it may be retarded greatly. In an intercurrent fever the sugar may be much lessened. In general the duration and the mildness increase with advancing age of onset. In the young there may be *acute cases* lasting a few weeks or months. In the middle-aged it may last ten years or more. Death may be due to cachexia, a complication, or intercurrent illness.

Treatment.—In general the patient should be freed from worry, should live in an equable climate, and take systematic exercise. The *diet: Allow* thin soups, meats (except liver), eggs, sour milk and its derivatives; such vegetable foods as salads, celery, cucumbers, tomatoes, and fruits (except sweet apples, peaches, or bananas); nuts, gluten flour; almond meal and bran bread. At times spirits or dry or acid wines may be allowed; also black coffee and tea. For sweetening, saccharin or perhaps levulose may be used. *Forbid* grains, roots, and tubers, sugars, starches, malt liquors, and sweet wine. *Drugs:* Many drugs are used. The more effective are codein, gr. ss–ij t. i. d.; potassium bromid, gr. xxx daily; arsenious acid or arsenic bromid; and antipyrin. Preparations of the pancreas are still on trial. With diacetic or β-oxybutyric acid in the urine give more carbohydrates, and give three or four ounces of sodium bicarbonate a day. In coma, inject intravenously sodium bicarbonate even up to ℥iij, and use oxygen inhalations and stimulants.

QUESTIONS.

Give the etiology of diabetes.

Discuss the relation of the pancreas to diabetes.

Give the symptoms of the ordinary case of diabetes.

Give the complications and describe diabetic coma.

Discuss the prognosis of diabetes.

Give its treatment.

DIABETES INSIPIDUS.

Etiology.—This is supposed to be a renal vasomotor paralysis of central or reflex origin. It is usually in males, before middle life, with a hereditary tendency; or in convalescence from acute infections. It may follow injury or diseases affecting the floor of the fourth ventricle, or perhaps the excessive drinking of cold water or alcoholic drinks.

Symptoms.—The onset is usually slow. There are great thirst and polyuria. The urine is colorless, perhaps 10 to 40 pints daily, with sp. gr. below 1005, with no albumin or casts. Rarely there is a trace of sugar. The daily output of urinary solids is nearly normal. The general health is usually good unless there is a brain tumor or similar condition. The polyuria may stop spontaneously, but is apt to last throughout life.

The **treatment** is unsatisfactory.

QUESTION.

Give the etiology and symptoms of diabetes insipidus.

RACHITIS (Rickets).

Etiology.—This is a disease of nutrition, and is doubtless due to a deficiency of fat and proteid, food, and perhaps to an excess of carbohydrates. It is usually seen in bottle-fed babies, or after too protracted nursing; in negroes and Italians; in city tenements with bad sanitation. It usually begins from the sixth to the sixteenth month.

Lesions.—The flat bones are thinner and flexible in places, elsewhere they may be thickened. The epiphyses of long bones are much enlarged; the epiphyseal line is broadened, there is an excess of cartilage; the bone throughout is deficient in lime salts, and consequently is flexible and often misshapen. Further details of the lesion are mentioned with the symptoms.

Symptoms.—The onset is usually insidious; the child is apt to have sweating of the head and restlessness at night, constipation, and to become pale and flabby; dentition is delayed, and the child learns to walk late. *Physical examina-*

tion: The *head* looks large; the veins are dilated; the forehead and the parietal and frontal bones are prominent; the occiput is flat, and the head looks quadrilateral. The sutures remain open for a year, the fontanels for even two or more years; hydrocephalus is rare; on the parietals and occipital bones there are spots that feel soft and crackle under pressure (craniotabes). *The trunk:* The rachitic rosary (a row of nodules at the chondrocostal junction) is present in most cases; it may disappear after four or five years. The chest is narrow and deep. There may be a deep depression at the lower end of the sternum (funnel breast); or in other cases a prominent sternum with vertical grooves on each side (pigeon breast); or Harrison's grooves, which extend laterally along the lower ribs. The free border of the ribs is everted, there is some kyphosis, the clavicle is thickened and more curved. There is a pot belly, and the liver and spleen are palpable from enlargement and displacement. There may be a flattened or figure-of-8 pelvis. *The limbs:* The lower ends of the radius, ulna, tibia, and fibula are enlarged and perhaps tender; the shafts of these bones are flexible, and there are often bow-legs or knock-knees, or in extreme cases very crooked legs. The humerus and femur are less often and less extensively involved. The ligaments may be lax, allowing abnormal movement. The legs are more deformed with attempts at walking. The growth in length of the bones is lessened, but ultimately they may become unusually strong and thick.

Complications.—Adenoids, large tonsils, large cervical nodes, a greater susceptibility to acute infections, especially bronchitis and pneumonia, attacks of diarrhea, convulsions, are all common. There may be laryngismus stridulus or tetany.

Course.—The active stage lasts until about the end of the second year. The deformities are more or less permanent, but often there are great physical and mental strength. Late rickets appearing from six to twelve years and congenital rickets have been described. Rickets does not kill, but the complications often do.

Treatment.—The diet should include meat, cream and other fat, and should be generous. Fresh air is important.

Drugs: Cod liver oil, phosphorus (gr. $\frac{1}{200}$ t. i. d.), calcium salts, iron, and tonics are recommended.

QUESTION.

Give the etiology, symptoms and physical signs, complications, prognosis, and treatment of rickets.

OBESITY.

Etiology.—There is often a family history of obesity, gout, or rheumatism. It may occur in children, but is more common after middle life and in women; with lack of exercise; an excess of food or of liquids, especially malt liquors; and is sometimes associated with anemia. The fat is derived from fatty and proteid food. When these are not fully oxidized the fat accumulates. When the more easily oxidized carbohydrates are eaten in excess there is deficient oxidation of proteids and fats and the development of obesity. This is the usual condition, but some cases occur in light eaters.

Lesions.—There is an excessive amount of fat in the subcutaneous tissue, in the omentum and subserous tissues, in the connective tissue of the viscera, and around the cells and fibers (fatty infiltration) of the liver, heart, etc. The blood may be plethoric, with an excess of red cells and hemoglobin (even 120 per cent.). Less often there is anemia.

Symptoms.—The patients may have firm tissues, but are short of breath, especially on exertion. Later, the heart weakens, becomes irregular, and often slow. Other persons may be anemic and have flabby tissues and a pasty skin. All cases stand acute fevers badly.

Treatment.—The diet should be restricted, the liquids should amount to about 30 ounces a day. Hearty eaters should eat less of all kinds of food. All should eat little sugar and starch. Too rapid or too radical changes in the diet weaken the patient and are bad. Exercise should be systematic and carefully regulated. A course at a spa is often very beneficial. Anemic cases need iron. Many special diets and methods of treatment are practised.

QUESTION.

Give the etiology, lesions, symptoms, and general treatment of obesity.

ADIPOSIS DOLOROSA (Dercum's Disease).

This is a rare condition, usually in middle-aged women. Large masses of subcutaneous fat develop on the limbs and trunk and are roughly symmetrical. The masses are tender and are the seat of neuralgic pains and paresthesias. In some cases the thyroid has been found atrophied. The tumors may be excised or thyroid extract may be administered.

DISEASES OF THE DIGESTIVE ORGANS.

APHTHOUS STOMATITIS.

THIS usually occurs in children, associated with fevers and indigestion. A little vesicle forms, and within a few hours changes to an ulcer that has a grayish adherent coating and is very tender. Aphthæ usually form on the inner surface of the lips and the cheeks, and the edge of the tongue. Salivation and a heavy breath are present. The ulcers last a week or more. The aphthæ should be touched with silver nitrate stick, and a potassium chlorate mouth-wash should be used.

ULCERATIVE STOMATITIS (Fetid Sore Mouth).

Sometimes this is contagious, occurring endemically or epidemically in institutions for children, in jails or barracks. A similar condition is due to mercury, lead, phosphorus or scurvy. Also it is found in persons who are cachectic and have dirty mouths. The edges of the gum are red, swollen, bleed easily, and show sloughs and ulcers that tend to surround the roots of the teeth. The teeth often loosen. The submaxillary lymph-nodes are swollen and tender. There are salivation, a fetid breath, painful mastication, a moderate fever, and sometimes an irregular skin rash. The condition may last a few weeks or become chronic.

Treatment.—Potassium chlorate is used as a mouth-wash, and also is swallowed in doses of ʒj daily. The contagious cases in institutions should be isolated.

PARASITIC STOMATITIS (Thrush).

The cause is Oïdium albicans, a yeast fungus; it usually occurs in poorly nourished babies with dirty mouths; and it is transmitted by dirty nipples, spoons, or fingers. In *adults it is* seen toward the end of wasting diseases. White spots appear on the tongue, grow and coalesce to form a grayish membrane. This can be brushed off, leaving a fairly normal mucous membrane, or in the severer cases a bleeding surface. The membrane is made up of filaments and cells of the fungus, and epithelium and mouth bacteria. It may spread beyond the tongue. The mouth is dry and sore and the saliva is acid. It lasts for weeks or months. Cure is difficult, but thrush is not found in mouths that are kept clean, therefore mouth-washes should be used in weak persons after every feeding. A mouth-wash of potassium chlorate or borax is used for the disease.

GANGRENOUS STOMATITIS (Noma; Cancrum Oris).

This is seen in children in poor general condition, especially after measles. It begins as an ulcer on the inner side of the cheek or on the lip or gums. The ulcer spreads rapidly, is ragged, has a black, sloughing base, with a gangrenous odor, and may eat away much of one cheek. There is a surrounding zone of brown, brawny tissue. Fever and the typhoid state are present; 80 or 90 per cent. die within a week or two. A few recover after a long convalescence with a scar that greatly disfigures the face. Septic lobar pneumonia or gangrene of the lungs is apt to develop. Noma may appear on the vulva or in the colon.

Treatment.—Noma can be prevented by careful hygiene of the mouth. When present, the base of the ulcer should be excised or cauterized with the thermocautery.

ACUTE GLOSSITIS.

In conditions of poor health or after exposure to cold and wet, with an abrasion of the tongue, infection may occur. The tongue becomes greatly swollen and may protrude. The protruding part is dry and cracked, the whole tongue is very painful and tender. It causes salivation, dysphagia, dysphasia, and dyspnœa with cyanosis. The related lymph-nodes are swollen and tender. There are fever and a rapid loss of weight and strength. Usually the inflammation increases for three or four days and then resolves rather rapidly.

Sometimes an abscess forms in one-half of the tongue, this ruptures, and after healing leaves a distorted tongue.

The **prognosis** usually is good.

Treatment.—Antiseptic mouth-washes and the constant sucking of ice. For the dyspnea, scarification of the tongue or even tracheotomy. Rectal feeding is sometimes necessary.

QUESTION.

Give the etiology, symptoms, and treatment of aphthous stomatitis, ulcerative stomatitis, thrush, noma, and acute glossitis.

FOLLICULAR TONSILLITIS.

This affection is more common in children and young adults who have a rheumatic tendency. It may be induced by irritants, such as tobacco or dust, or by exposure to cold and wet. One attack predisposes to other acute attacks and to chronic tonsillar enlargement. Streptococci or staphylococci of suppuration are usually found on the tonsil.

Symptoms.—There is usually an abrupt onset of fever (104° F. or higher), with marked aching and prostration. The fever goes by lysis within a week. Locally the tonsils are swollen, red, and have whitish cheesy plugs protruding from the crypts. These may spread over the surface of the tonsil like a false membrane, but are easily brushed off, leaving an intact surface. There is pain in the throat, especially upon swallowing or speaking.

Treatment.—A mixture containing tincture of the chlorid of iron, potassium chlorate, and glycerin often helps. The rheumatic cases should take salicylates. Locally, sprays, mouth-washes, and gargles may be used.

QUESTION.

Give the etiology, symptoms, and treatment of follicular tonsillitis.

SUPPURATIVE TONSILLITIS
(Peritonsillitis; Quinsy Sore Throat).

This condition usually begins as a follicular tonsillitis. The throat symptoms become more marked, the voice very thick and "throaty"; there may be dyspnea and some coughing. The throat is covered with thick tenacious mucus; one or both tonsils are much swollen, reddened, and pushed toward the middle line; the soft palate and uvula are red, swollen, and edematous. The swelling and pain increase for a few days, then an abscess forms and ruptures, usually into the mouth, discharging fetid pus. After this the pain and general symptoms lessen rapidly, and in a few days the sinus heals. If both tonsils are involved, one is usually attacked a day or two after the other. The whole illness lasts a week or ten days. Resolution without abscess formation sometimes occurs.

Treatment.—When the abscess points it should be incised.

QUESTION.

Give the etiology, lesions, symptoms, and treatment of quinsy.

ADENOIDS AND HYPERTROPHIED TONSILS.

Etiology.—These are common in childhood. There may be an hereditary tendency or they may follow repeated acute attacks of pharyngitis or tonsillitis. Either may occur alone.

Lesions.—The tonsils show hyperplasia, but later fibrous tissue predominates, and they become hard and smaller. In the marked cases the tonsils almost reach the middle line of the fauces. The adenoids form projecting lobular tumors attached to the roof or back of the nasopharynx; sometimes they are pedunculated.

Symptoms.—The tonsils are large, protruding, and may be soft or hard. The adenoids may possibly be seen through the nose, and are readily felt by the finger in the mouth. They bleed easily. The child has a chronic catarrh, with acute exacerbations of the nose, nasopharynx, often of the Eustachian tubes and middle ear, of the larynx, and bronchi. There are frequent attacks of acute tonsillitis. There is mouth-breathing, especially at night, with snoring and disturbed sleep. In time a characteristic facies may develop with a stupid look, open mouth, large lips, and a narrow jaw. There is a tendency to asthma, spasm of the larynx, nightmares, and mental dulness. There may be malnutrition and anemia. The child is better in summer and worse in cold or damp weather. The lesions and the symptoms get worse for a year or two, are nearly stationary until puberty, and then becomes less troublesome.

The **treatment** is the excision of the tonsils and adenoids, and the use of such tonics as cod-liver oil and the syrup of the iodid of iron.

QUESTION.

Give the etiology, symptoms, and treatment of enlarged tonsils and adenoids.

RETROPHARYNGEAL ABSCESS.

This occurs in children; it is often primary, but may be secondary to scarlatina or other infections. There may be a moderate fever. After two or three days there are dysphagia, dyspnea, with signs of marked obstruction in the throat, and a thick voice. Inspection shows a red swelling in the posterior wall of the pharynx. At first this is hard, but later softens. In a few days the abscess ruptures. The pus may enter the larynx and lead to asphyxia or be followed by pneumonia.

The **diagnosis** is made by feeling the abscess with the finger in the pharynx.

The **treatment** is incision.

QUESTION.

Give the etiology, symptoms, and treatment of retropharyngeal abscess.

ANGINA LUDOVICI (Ludwig's Angina).

This is secondary to scarlatina, diphtheria, trauma, or a wound of the mouth. The lesion is a cellulitis of the tissues of the floor of the mouth, often with gangrene, and usually due to Streptococcus pyogenes alone or with other bacteria.

The **symptoms** begin abruptly with high fever. Locally there is a very marked and rapid swelling of the submaxillary tissues and the neck, with difficulty in breathing, swallowing, and speaking, and with great pain. The prostration is marked. Death often follows in a few days.

The **treatment** is surgical and must be prompt.

QUESTION.

Give the etiology, symptoms, and treatment of Ludwig's angina.

STRICTURE OF THE ESOPHAGUS.

Etiology.—It usually is due to carcinoma of the esophagus, less often to cicatricial contraction or to pressure from without. It is usually near the pharynx or the cardia.

The **symptoms** are an increasing difficulty in swallowing, shown first with solids, but later with liquids also; the food regurgitates. A dilatation above the stricture usually develops. There is progressive emaciation from starvation.

Physical Signs.—If one listens at the left of the lower thoracic spine, when the patient swallows, the normal sound is modified, absent, or much delayed. The esophageal bougie may determine the site and the size of the stricture, but it should be used with care.

The **treatment** consists of gradual dilatation with the bougie, liquid feeding, and later rectal feeding, or gastrostomy.

DILATATION OF THE ESOPHAGUS.

This occurs above a stricture.

Symptoms.—From a few minutes to three or four hours after eating food is regurgitated. It is alkaline, often putrid, and contains mucus, pus, or blood. The symptoms of the stricture also are present.

CARCINOMA OF THE ESOPHAGUS.

This usually occurs in males and after the fortieth year. The growth is an epithelioma that encircles the esophagus and is usually in the lower third. It may be hard or soft, and often ulcerates. The cervical nodes may be enlarged.

The **symptoms** are those of stricture and dilatation of the esophagus and of cancer. The regurgitated food contains mucus, blood, and pus, with perhaps bits of the tumor.

QUESTIONS.

Give the etiology, symptoms, and treatment of stricture of the esophagus.

Give the etiology and symptoms of dilatation of the esophagus.

Give the etiology, pathology, and symptoms of cancer of the esophagus.

DISEASES OF THE STOMACH.

Test-meals.—*Ewald's test-breakfast* consists of a roll and 8 ounces of water or tea without milk; it is withdrawn after one hour. *Boas' test-breakfast* consists of strained oatmeal gruel made with water (it contains no lactic acid); it is withdrawn after one hour. The *Leube-Riegel test-dinner* consists of soup, meat, and a potato or roll; it is withdrawn after three and a half to four hours.

The **stomach-tube** is a flexible rubber tube about 5 feet long and $\frac{3}{8}$ inch in diameter, fitted with a funnel in one end. To pass the tube the patient sits erect with mouth slightly open; the wetted tube is passed to the pharynx, and then is swallowed and gently pushed into the stomach. The cardiac end of the stomach is about 18 inches from the incisor teeth. The *contents of the stomach are withdrawn* by the suction of a rubber bulb or by syphonage, aided by pressure on the epigastrium and voluntary contraction of the abdominal muscles. For *lavage*, water is poured into the tube through the funnel until the stomach feels full; the funnel is then lowered and the water syphoned out. This process is repeated several times until the stomach is clean. The liquid used is warm water or a solution of common salt, sodium bicarbonate, boric or salicylic acid, or other drugs. The details of the examination of the stomach-contents can not be given here.

The *motor function* is examined with the help of the stomach-tube. If the motor function is not deficient, the stomach will be empty six or seven hours after the Leube meal, or two and a half hours after the test-breakfasts.

The *absorbing power* of the stomach is sometimes tested by giving potassium iodid gr. ij–v in a capsule, and testing the saliva for iodin with starch-paper and acid every five minutes. Normally iodin is found in the saliva within fifteen minutes.

ACUTE GASTRITIS.

Etiology.—A predisposition exists in certain persons, especially the poorly nourished, the gouty, and those in unhygienic surroundings. The exciting cause may be too much food or bad food, alcohol, dirty nipples or nursing-bottles, irritating drugs, perhaps cold, and various micro-organisms. Acute gastritis may accompany certain infectious diseases, such as influenza, scarlet fever, or measles.

The **lesion** is an acute catarrh (see p. 18).

Symptoms.—A few minutes or hours after eating there are a feeling of epigastric weight and fulness, dull pain or cramps, nausea, eructation, and vomiting, which may be frequent. The vomitus contains the food but little digested, often fermented and sour, little mucus, little or no hydrochloric acid, often some fatty acids, and not rarely bile. The tongue is coated, the breath heavy, the epigastrium tender. There may be constipation in an adult, but diarrhea is usual in infants. Mild cases are afebrile. Severe cases have a temperature of 101°–103° F. and febrile symptoms. Mild cases last but a few hours, leaving some epigastric distress for a few days. Severer cases last three or four days, and then subside gradually.

The **prognosis** of an attack is good, but the tendency to attacks persists, and they may become more protracted.

Treatment.—The stomach should be emptied by vomiting, induced, if necessary, after filling the stomach with water. Calomel, gr. j–iij, should be given; morphin hypodermically may be needed. Counterirritation on the epigastrium is used. For a day the diet should consist of water and soda only; then milk or other liquid may be added, and in convalescence the ordinary diet may be gradually resumed. The patient should stay in bed until the symptoms subside.

TOXIC GASTRITIS.

This is due to irritant and corrosive poison, such as arsenic, phosphorus, mercury bichlorid, carbolic acid, and strong acids or alkalies.

Lesions are present in the mouth, esophagus, stomach, and often in the intestine. There is intense congestion with bleeding into the mucous membrane and degeneration and desquamation of the epithelium. With corrosive poisons there are necrosis and sloughing of the mucous membranes or deeper tissues, or possibly a perforation.

Symptoms.—A prompt and sudden onset of burning pain from the lips to the epigastrium, dysphagia, and repeated vomiting. The vomitus contains the poison, mucus, blood, food, possibly sloughs and pieces of mucous membrane. The mouth and throat are red or covered with a slough that varies with the poison; the abdomen is distended; the epigastrium very tender. Later there is often diarrhea, with frequent, small mucous and bloody stools, tenemus, and colic. There may be collapse, fever, and petechiæ. The urine is decreased, albuminous, and often bloody. The severer cases have restlessness, convulsions, stupor, and coma. Death may follow in a few hours with collapse, or after a few days from exhaustion or uremia. Many recover, but a cicatricial stricture of the esophagus or stomach, or gastric ulcer or atony may follow.

The **prognosis** depends upon the nature and amount of the poison and the time that it remains in the stomach.

Treatment.—The proper antidotes should be used. The general treatment consists of lavage, demulcent drinks, rectal feeding, stimulation, and morphin for the pain.

MEMBRANOUS GASTRITIS.

This is a terminal infection of infectious fevers, and is diagnosed at autopsy.

PHLEGMONOUS GASTRITIS.

This affection may occur in pyemia, after injury, or without discoverable cause. In the submucous coat there is pus, either as a localized abscess or diffuse phlegmon.

The **symptoms** are those of intense gastritis and general sepsis. The localized form may give a tumor that may be made out by physical examination. The abscess may rupture into the stomach with recovery. The diffuse form is fatal, usually within a week.

QUESTIONS.

What are Ewald's test-breakfast, Boas' test-breakfast, and the Leube-Riegel test-dinner, and how are they used?

Give the technic of lavage.

How is the motor function of the stomach tested?

Give the etiology, lesions, symptoms, and treatment of acute gastritis, of toxic gastritis, of phlegmonous gastritis.

DILATATION OF THE STOMACH (Gastrectasia).

Etiology.—Acute dilatation is rare; it may follow acute fevers or rapid ingestion of a large quantity of liquid. Chronic dilatation is common, usually in late middle life. Its causes are (*a*) pyloric obstruction from cancer, scar-tissue, congenital stenosis, a kinking from adhesions, or pressure from outside; (*b*) muscular atony from repeated overdistention; (*c*) chronic gastritis; (*d*) cachectic states.

Lesion.—The stomach holds more than three pints, perhaps many quarts, and there is chronic catarrh.

Symptoms.—Food accumulates in the stomach and ferments; absorption is defective. There are eructations, a sense of weight in the abdomen, and every few days the vomiting of several pints of foul-smelling sour matter that contains remnants of several meals. *Microscopically*, the vomitus contains food remnants, bacteria, sarcinæ, yeast, and mucus. *Chemically*, there are the organic acids, and sometimes hydrogen sulphid; hydrochloric acid is usually diminished, but may be normal or increased; digestion is defective. *Physical examination:* On inspection when the patient is erect the outlines of the stomach may be seen, especially after its distention with gas, and peristalsis may be visible. On palpation the outline of the stomach can often be made out, sometimes the hard pylorus can be felt; "splashing" is marked. On percussion the area of gastric tympany is much increased in both vertical and transverse diameters; the greater curvature is below the navel. When the patient is erect and the stomach contains liquid, there may be a band of dulness at the lower part of the stomach that shifts with a change of position. Auscultatory percussion is most accurate. *Other symptoms* are emaciation, anemia, constipation, scanty urine, insomnia, syncope, or perhaps tetany.

The **prognosis** depends largely on the cause and is usually bad.

Treatment.—Lavage daily until fermentation stops, with boric or salicylic acid solution; later with warm water.

Diet: Frequent small meals of scraped beef, meat juice, eggs, and nutritive liquids. Liquids may be given by rectum for the thirst. Surgery may help.

QUESTION.

Give the causes, symptoms, and treatment of gastrectasia.

CHRONIC GASTRITIS.

Etiology.—(1) *Dietetic causes:* Excessive eating, improper food, irregular meals, deficient mastication. Tea, coffee, alcohol, tobacco, drugs. (2) *General causes:* Anemia, tuberculosis, gout, diabetes, Bright's disease, protracted diseases of the stomach, portal obstruction.

Lesions.—(1) *Simple* and (2) *mucous gastritis* are chronic catarrh (p. 18), most marked near the pylorus. The mucous membrane may be polypous, pigmented, ecchymotic, or ulcerated. There is an excess of mucus. *Microscopically*, the acid and other cells look alike; the glands are irregularly branched, and some are cystic. The stomach often is somewhat dilated. (3) *Sclerotic form:* The mucous membrane is smooth, contains few or no glands, and an excess of connective tissue. The wall may be thin (phthisis ventriculi); or thick and firm with great contraction of the stomach (cirrhosis ventriculi).

Symptoms.—The onset is slow, and at first the course is marked by exacerbations and remissions. The ordinary symptoms are a variable loss of appetite; nausea, and vomiting of a little mucus in the morning, or of sour-smelling food and mucus after meals; gastric distress, pyrosis, eructations after meals; flatulence and constipation; palpitation; vertigo, headache, physical and mental indisposition, low spirits, and a moderate loss of weight. The urine is scanty. In *simple catarrhal gastritis*, the motor function is somewhat deficient, but the stomach usually is empty in the morning. After a *test-breakfast* the stomach-contents are acid to litmus, HCl is usually diminished; mucus and possibly a little lactic acid are present. In *mucous gastritis* there is no HCl, pro-enzymes *are present,* mucus is abundant. In *atrophic* or *sclerotic gas-*

tritis the motor function is normal or exaggerated, HCl, proenzymes, fatty acids, and mucus are absent; the stomach may be felt as a vague tumor; anemia is marked. *Acid gastritis* is a form with excess of HCl.

Prognosis.—It varies with the removability of the cause and the duration and extent of the lesions. Mild cases may become practically well, others much alleviated.

Treatment.—Most important is the elimination of the cause. The diet should be milk or digested food only for two to four weeks; later a little meat may be added; then thoroughly boiled starch, etc. Alcohol, tea, and coffee should not be given. The stomach should be cleaned by lavage daily, or in the mild cases by drinking hot water before meals. General hygienic measures help. *Drugs:* Laxatives when necessary; HCl ♏xx–lx after meals, perhaps with pepsin. Bitter tonics should be used only after the stomach is secreting fairly well.

QUESTION.

Give the etiology, varieties, lesions, symptoms, and treatment of chronic gastritis.

PEPTIC ULCER OF STOMACH AND DUODENUM.

Etiology.—*Gastric ulcer* is more common in women from twenty to forty, in servants, in those whose occupations involve pressure upon the stomach, and in anemia. Trauma to the epigastrium may cause it. *Duodenal ulcer* is more common in males, and may follow extensive superficial burns. Pathologically an ulcer may be due to embolism with digestion of the infarct, to trauma of the mucous membrane, bacterial necrosis, or possibly excessive HCl.

Lesions.—The ulcer is usually single, and occurs most frequently near the lesser curvature, on the posterior wall or at the pylorus. Acute ulcers are round and punched out, the edge is soft and the floor smooth. Chronic ulcers have a rounded thick edge. The ulcers are of all sizes, perhaps 1 inch is an average diameter. Small ulcers may involve the mucosa only, larger ones may erode the muscularis. Perforation is not rare. After months a scar may form, which may later cause pyloric stenosis, hour-glass stomach, or other deformity. Perforation may result in general or localized peritonitis, subphrenic abscess, or subphrenic pyopneumothorax. An eroded vessel may be seen in the floor of the ulcer after bleeding.

Symptoms.—The chief symptoms of the typical cases are (1) *vomiting*, associated with more or less indigestion, usually occurring an hour or two after meals. The vomitus contains an excess of HCl. (2) *Pain* in the epigastrium, often intense and gnawing, usually less when the stomach is empty. It occurs also as intense paroxysms of epigastric pain radiating to the back and sides, lasting an hour or two. (3) *Hemorrhage*, usually profuse and promptly vomited; the vomited blood is acid, bright red, and not frothy. If vomiting is delayed, the blood may look like coffee-grounds. Tarry stools follow. A large hemorrhage causes general symptoms, such as syncope, convulsions, etc.; repeated small hemorrhages cause profound anemia. (4) *On examination* the epigastrium is tender, and there may be a tender spot in the back to the left of the lower thoracic spines. An indefinite mass may sometimes be felt in the epigastrium or right hypochondrium.

Course.—There are *latent cases* that give no symptoms. The *usual cases* have the above symptoms, last several weeks, may lose some weight and strength, and become anemic, but recover. Recurrences are common. Rarely hemorrhage or perforation may be the first thing noticed. The patient may die from bleeding or perforation, or from exhaustion. After healing there may be symptoms from contraction of the scar, or cancer may develop in the scar.

Duodenal ulcer has similar symptoms, but the pain may be in the right hypochondrium and is apt to come on several hours after meals. There may be tarry stools without hematemesis. The diagnosis is very difficult.

Treatment.—Rest in bed for many weeks. A liquid diet for two or three weeks. Nutritive enemata may be needed in some cases. *Drugs:* Carlsbad salt, ℥j every morning; bismuth, ʒj–ij daily; silver nitrate, gr. ¼ t. i. d. Opium for the pain may be necessary. Surgical treatment is indicated in perforation, repeated hemorrhage, a chronic ulcer, not yielding to medical treatment, and for the sequels, such as pyloric stenosis.

QUESTIONS.

What are the etiology, lesions, symptoms, complications, and treatment of ulcer of the stomach?

What special symptoms suggest ulcer of the duodenum?

CARCINOMA OF THE STOMACH.

Etiology.—It is usually primary; it occurs after forty, more commonly in males. It may follow chronic gastritis or ulcer of the stomach, or an hereditary tendency.

Lesions.—It is usually at the pylorus, less often on the lesser curvature or at the cardia. It may be adenocarcinoma, medullary, scirrhous, or colloid cancer. It may be a diffuse growth, a cauliflower growth, or a thick ring at the pylorus. It often ulcerates. There is usually chronic catarrh. With cancer at the cardia, the stomach is small and the esophagus dilated; at the pylorus, the stomach is dilated. Metastases are found in the lymph-nodes of the abdomen, also of the neck and groin; in the liver, peritoneum, skin, etc.

Symptoms.—After gastric catarrh or general failure of health for a variable time the patient is apt to have: (1) *pain* in the stomach and back, steady and dragging; less often severe paroxysms occur. (2) *Vomiting* of undigested food. (3) *Bleeding* into the stomach; this is usually an oozing and the blood appears in the vomitus as coffee-ground matter. (4) *Examination* of the patient; on *inspection* there may be a fulness or a definite tumor in the epigastrium or visible peristalsis. On *palpation*, in the epigastrium or to the right of it there is felt a hard nodular tender tumor that moves with respiration, with filling of the stomach and with manipulation; later it may be adherent; it often transmits the aortic pulsation very distinctly. The signs of a dilated stomach may be present. (5) *The stomach-contents:* After a test-breakfast the contents are acid, contain undigested food, HCl is absent or much diminished, lactic and fatty acids are abundant, ferments are often absent. Microscopically there are the Boas-Oppler bacillus, sarcinæ, blood-pigment or red cells, pus-cells, and possibly little masses of the tumor. The coffee-ground matter of the vomitus shows pigment masses, and sometimes remnants of red cells.

The other symptoms are often those of dilatation, constipation, tarry stools; attacks of diarrhea are common late in the disease; the blood shows a very severe secondary anemia, or sometimes resembles that of pernicious anemia; fever is inconstant. The urine contains indican in excess. Cachexia develops.

Complications.—Metastases, perforation with general peritonitis or local peritonitis; rarely sloughing or gangrene of the tumor; the terminal infections.

Course and Prognosis.—There may be periods of improvement, but the disease is usually fatal in about a year.

Varieties.—(1) *Latent cases:* Cancer may be found at autopsy in cases that have given no stomach symptoms. (2) The stomach symptoms may be completely masked by those of secondary growths in the liver. (3) There may be progressive asthenia without stomach symptoms, resembling pernicious anemia. (4) A cancer in the scar of an old ulcer may be associated with an excess of HCl.

Treatment.—The chronic catarrh and dilatation, the pain and bleeding need treatment. Surgery is often indicated.

QUESTION.

Give the etiology, lesions, symptoms, prognosis, and treatment of cancer of the stomach.

HEMATEMESIS.

Etiology.—(1) Bleeding into the stomach may come from (*a*) *an eroded vessel*; in cancer, ulcer, arteriosclerosis with miliary aneurysms, varicose veins, trauma (from stomach-tube, etc.), corrosive poison, erosions following operations on other abdominal viscera, or the rupture into the stomach of an abdominal aneurysm. (*b*) From *congestion*—in acute gastritis or perhaps vicarious menstruation; portal obstruction, or splenic enlargement. (*c*) General diseases, such as variola, measles, yellow fever, acute yellow atrophy, phosphorus-poisoning, severe anemia, severe jaundice, hemophilia, purpura, scurvy, etc. (*d*) In hysteria, epilepsy, general paralysis, or without discoverable cause. (2) The blood may come from the nose, throat, lungs, or the esophagus, and be swallowed and later vomited.

An eroded vessel can usually be found at autopsy, but in the other cases there may be no discoverable lesion of the stomach-wall.

Symptoms.—There may be the general symptoms of a large hemorrhage, such as dizziness, faintness, syncope, sudden pallor. This may be followed by hematemesis, and later by tarry stools. There may be repeated small bleedings, with a more gradual development of anemia. With *hematemesis* there is the history of some cause; the blood is brought up with vomiting, but a cough may follow; the blood is usually clotted, often in large amounts, bright or dark red, or partly digested, looking like coffee-grounds; it is usually acid. *Microscopically,* it contains red cells or their remnants, and often particles of food. Later the stools are tarry.

Treatment.—Rest in bed. Cracked ice, astringents, especially suprarenal extract, may be swallowed. Morphin hypodermically. When the bleeding stops, saline infusions or enemata are given. Rectal feeding for a few days is needed. Afterward, tonics and iron. If the bleeding is rather profuse and persistent, the limbs may be tied off successively to prevent venous return. An operation may find the bleeding point.

QUESTIONS.

Give the etiology of hematemesis.
Give the diagnostic points of blood that has been vomited.
What is the treatment?

GASTRIC NEUROSES.

Etiology.—These are associated with neurasthenia, hysteria, or other nervous states.

The **symptoms** may involve any function of the stomach.

The most important indication for **treatment** is the causative condition. The more important neuroses and some special forms of stomach treatment are the following:

(1) **Motor Neuroses**: (*a*) In *supermotility* the stomach is emptied too soon. This is usually associated with an excess of HCl or of gastric juice. It is diagnosed by the stomach-tube. (*b*) *Peristaltic unrest* is common; there is gurgling after eating, with much mental distress; it may involve the intestine also, and there may be retroperistalsis. (c) Nervous

eructations are common; usually the gas is air that has been swallowed. (*d*) *Nervous vomiting* is rather common; it usually occurs after meals, often without nausea; nutrition may not be affected. (*e*) *Spasm of the pylorus* usually occurs with excess of HCl or gastric juice. If persistent enough, dilatation and atony may follow. *Treatment:* Small meals eaten slowly; belladonna or codein. (*f*) *Pyloric insufficiency* allows the passage of fluids in both directions through the pylorus.

(2) **Sensory Neuroses:** (*a*) *Gastralgia* is important and not very rare; clinically it resembles gastric crises in tabes and the pain of ulcer of the stomach. It is diagnosed by exclusion and the existence of the nervous cause. *Treatment:* Counterirritation to the epigastrium; chloroform or compound spirit of ether by mouth. Morphin hypodermically may be needed. (*b*) *Hyperesthesia* usually occurs during digestion, and results in a sense of fulness, weight and burning in the epigastrium; it may affect the general nutrition. *Treatment:* Liquid diet for a week or two, silver nitrate before meals, and perhaps bromids for two or three months. (*c*) *Nervous anorexia* may lead to an extreme aversion to all food, emaciation, and restlessness. Weir Mitchell treatment is indicated.

(3) **Secretory Neuroses:** (*a*) *Hyperchlorhydria:* The *percentage of HCl* during digestion is increased. The symptoms are inconstant, but there may be burning or pain in the stomach about two hours after meals, which may be relieved by proteid food, water, or alkalies. Nutrition is seldom affected. *Treatment:* Large doses of alkalies one and two hours after meals; much proteid food. (*b*) *Supersecretion* is an excess of the *total amount of gastric juice;* it may be continuous or intermittent, and is usually associated with hyperacidity. There may be burning in the stomach, vomiting of much burning liquid, with or without food remnants. *Treatment:* Lavage with astringent solutions may help. (*c*) *Subacidity* is marked by decrease or absence of HCl. Enzymes or pro-enzymes are usually present. (*d*) *Achylia gastrica nervosa* is an absence of all gastric secretion. If the motor functions are unimpaired, special stomach symptoms are usually absent. Lactic acid is not present. The

condition may last for years. *Treatment:* Less proteid food. HCl after meals.

The **prognosis** of all forms is that of the general nervous state. Sooner or later they usually disappear.

QUESTIONS.

Give the etiology of gastric neuroses.
Mention the more important ones with their symptoms.

DISEASES OF THE INTESTINES.

CATARRHAL ENTERITIS.

Etiology.—*Primary cases* are due to (*a*) poisons, such as ptomains, arsenic, mercury, irritating cathartics, strong alkalies or acids. (*b*) Abnormal fermentations in the intestine, due to absence of bile or other causes. (*c*) Other primary cases occur after excessive or improper food, certain drinking water in persons unaccustomed to it, or impure water. In relation to waters and foods, idiosyncrasy is important. (*d*) Chilling of the skin may cause it. (*e*) Bacteria, probably the colon bacillus and others, may be the real cause, but this is not definitely understood. *Secondary cases* occur with (*a*) peritonitis, hernia, intestinal ulcers; (*b*) with chronic congestion from portal obstruction; (*c*) with acute infections, such as typhoid, dysentery, cholera, sometimes tuberculosis, pneumonia, etc.; (*e*) with cachexias of anemia, cancer, nephritis, or other conditions.

Lesions.—The *acute form* is a typical catarrh, except that congestion is rarely seen post mortem, and the lymphoid tissue often is swollen. The lesion may involve any part or the whole length of the large and small gut. *Chronic forms* show the lesions of catarrh, usually with increase of connective tissue and thickening, and (with chronic congestion) a slaty pigmentation of the villi and follicles. In *children* the mucous membrane may be thinned.

Symptoms.—*Acute cases:* The symptoms of gastritis are often associated. The *enteritis* causes intestinal colic, borborygmi, more or less tenderness, tympanites, and diarrhea.

The *stools* at first are large and fecal, but later become softer, watery, yellowish or greenish, and very offensive. Microscopically they show red and white blood-cells, epithelium, mucus, many bacteria, and food remnants. There may be from three to a dozen or more daily. Other symptoms are increasing weakness and sometimes fever for a few days. Some cases have marked *toxemic* symptoms with fever, aching bones and muscles, marked prostration, much indican and perhaps albumin in the urine.

Varieties.—(1) Steatorrhea (fatty diarrhea) may be due to deficient pancreatic secretion. The stools contain an excess of fat which at times is visible to the naked eye. (2) Emotion and sometimes irritating food may cause a diarrhea from increased peristalsis without actual inflammation. (3) With catarrh of the duodenum only there may be no diarrhea; gastritis is usually present; there are local pain and tenderness in the epigastrium and often jaundice. (4) Catarrh of the jejunum and ileum is marked by gurgling, distention, and colic in the middle and lower abdomen. The stools may be formed and contain white or bile-stained flakes of mucus intimately mixed with fecal matter. Much undigested food may be present (lienteric diarrhea). (5) With catarrh of the upper part of the colon the stools are large, fecal, not very frequent, and contain mucus on the outside or more or less intimately mixed. (6) With catarrh of the rectum (proctitis) the stools are very frequent, very small, mucous, often bloody and without fecal matter. There is tenesmus.

Course.—The diarrhea usually disappears slowly after two days or a week. There may be a tendency to recurrence, or it may become chronic.

Chronic enteritis is marked by chronic diarrhea, colic, loss of weight and strength. It may alternate with periods of constipation. With chronic congestion and in the terminal stages of cachexia there may be no pain.

Treatment in Adults.—*Acute diarrhea:* Rest in bed, a liquid diet. If seen early, the bowels should be emptied by castor oil or calomel. After a few hours bismuth, gr. x–xxx every two or three hours, may be given. Intestinal antiseptics or morphin may be added. Counterirritation may relieve the colic. An enema of 3 or 4 ounces of starch-water with tincture of opium, ♏xx every six hours, may relieve tenesmus. With toxemic symptoms the colon should be irrigated. The *chronic* cases during an exacerbation are treated like acute cases. A change of climate often cures them.

DIARRHEAS OF CHILDREN.

Etiology.—One of the varieties of Bacillus dysenteriæ is usually present. Diarrhea is common in bottle-fed babies during the first or second summer, especially in dirty surroundings and with bad air.

The **lesion** is a catarrh of the small and large intestines with swollen or ulcerated follicles. There may be croupous inflammation of the ileum and colon. The fatal cases often have bronchopneumonia and congested kidneys. The spleen is large; the brain anemic.

Symptoms.—(1) *Acute dyspeptic diarrhea* may follow a few days of slight indigestion, or begin suddenly, perhaps with a convulsion. The child vomits, and after a few hours has colic and diarrhea. The *stools* become more liquid but are fecal, and may be grayish or green, sometimes frothy and very offensive. The fever and toxic symptoms vary much, but the temperature may be 104° F. It usually clears up in two or three days, but in a weak child may be fatal. It often marks the invasion of an acute infectious disease.

(2) *Cholera infantum:* The onset is abrupt, often with a convulsion. There are a coated tongue, incessant vomiting, colic. The *stools* at first are diarrheal but after a few hours very frequent, watery, with little smell. Collapse, high fever (105° F.), a very rapid, feeble pulse (160–200), and great restlessness are present. After a day the *loss of liquids* is shown by a cold ash-gray skin, depressed fontanel, shrivelled tissues, intense thirst, a red dry tongue, and perhaps suppression of the urine; the stools often decrease in number and amount; there are retraction of the head, stupor, and convulsions. The child usually dies in collapse within a few days, but in some cases the symptoms clear up after a few days, and the child convalesces slowly.

(3) *Acute ileocolitis* may follow dyspeptic diarrhea in

summer or complicate any acute infectious disease at any season. *Symptoms:* After a day or two of ordinary diarrhea, the vomiting stops, but the colic increases, the *stools* are frequent and large, contain more mucus and usually blood; there is great tympanites and the colon is tender. The fever remains high, the prostration and loss of weight are severe. The fever may abate after a few days, but the diarrhea often persists for a few weeks, when convalescence may begin. Many cases die from exhaustion in three or four days or in an exacerbation of the less acute forms or from bronchopneumonia. This is a serious disease.

Treatment.—In summer, if the diarrhea does not improve within two or three days, the child should be sent to the country and kept out of doors all day in bed. Baths at 90° F. for five minutes relieve the temperature and the restlessness. *The Diet:* In mild cases food may be given less frequently, in smaller amounts, and more diluted. The severer cases should have only boiled water, barley-water, or egg-water every two hours. Lavage relieves the vomiting. With frequent stools or marked toxemia the colon should be irrigated two or three times on the first day and once a day afterward. *Drugs:* Calomel or castor oil are given at the onset. Afterward, bismuth, about a dram a day to a child of one year. Camphor, chloroform, or in an older child a little morphin may be given for the pain. Stimulation often is needed. For cholera infantum salt solution, 8 ounces or more, should be given twice a day by hypodermoclysis; morphin sulphate, gr. $\frac{1}{100}$, and atropin sulphate, gr. $\frac{1}{80}$, may be given hypodermically to a child of one year for the pain. Antidysenteric serum has given very promising results in many infantile diarrheas.

Celiac disease is a chronic diarrhea in children from one to five years of age. The *stools* are large, frothy, very foul, and gruel-like. The abdomen is distended and boggy. Anemia, weakness, and emaciation are progressive, and death usually follows.

QUESTIONS.

Give the etiology, the lesions, and the symptoms of acute and chronic enteritis in adults.

Give the principal varieties.

What is the treatment?

Give the etiology, lesions, varieties, symptoms, prognosis, and treatment of the diarrheas of children.

Ulcerative colitis is usually found in middle-aged or older men. The colon usually is dilated and thickened, but sometimes it is contracted. There are many ulcers in the mucous membrane, and between them the mucosa is dark in color and may be polypous. The onset is insidious. There are attacks of lienteric diarrhea, rarely with pus or blood in the stools. In the intervals there is constipation. Loss of flesh and strength is marked, the disease usually lasts for years, and the patient may die of exhaustion or perforation.

Other ulcers of the intestine: (1) Follicular ulcers are very common in children. (2) Stercoral ulcers are found in old persons in the pouches of the colon that contain scybala. (3) Solitary ulcer in the colon or cecum is seen rarely. (4) Ulcers occur with carcinoma, sarcoma, tuberculosis, syphilis of the colon, and in typhoid and dysentery. (5) They may be due to perforation, *from* the peritoneum *into* the gut in tuberculous peritonitis or local peritoneal abscess, especially at the appendix. With ulcers of the colon there is usually diarrhea. The *stools* may contain pus, shreds of tissue, mucus, especially in sago-like masses, and there may be bleeding. There is usually pain, either localized at the site of the ulcer or a more general colic. Perforation may occur with general peritonitis, or abscess behind the peritoneum. Ulcers in the rectum may be seen by means of Kelly's tubes.

QUESTION.

Give the causes of ulcers of the colon and the diagnosis.

APPENDICITIS.

Etiology.—*Bacteria:* Bacillus coli communis, Streptococcus pyogenes, Staphylococcus pyogenes aureus, B. proteus, B. typhosus, and B. tuberculosis have been given as causes. *Other points:* (1) Obstruction to the lumen of the appendix: (*a*) congenital from unusual length, site, or arrangement of meso-appendix; (*b*) from stricture near the cecal end; (*c*) peritoneal adhesions; (*d*) fecal concretions or foreign bodies. (2) *Other infections:* Typhoid, tuberculosis, actinomycosis. Appendicitis is more common in males, from youth to middle life, and may follow muscular strain, trauma to the abdomen, or errors of diet.

Lesions.—(1) *Simple catarrhal or obliterative appendicitis*—an exudative inflammation usually of all coats of the appendix. There is a tendency to complete or partial obliteration of the lumen. A stricture may form and beyond it there may be a dilatation, filled with serum or pus or forming a cyst. Recurrent attacks are common. (2) *Ulcerative appendicitis:* The mucous membrane ulcerates. The ulcers may heal and perhaps form strictures, or may perforate. (3) *Acute infective appendicitis, interstitial or parietal,* follows the previous forms. There is necrosis or gangrene. This may be limited (with a small perforation); or annular, cutting off the rest of the appendix; or the whole appendix may be gangrenous. *Perforation* may be followed by general septic peritonitis, or by localized peritoneal abscess, walled off by adhesions, containing fetid pus. Behind the peritoneum it may burrow extensively. Either intraperitoneal or extraperitoneal abscesses may rupture into the general peritoneal cavity, the ileum or large intestine, the bladder or elsewhere.

Symptoms.—In some cases the *onset* is slow with anorexia, constipation, rather indefinite abdominal pain, perhaps slight fever and indisposition. Usually it is abrupt with anorexia; nausea and vomiting for about a day, constipation, abdominal pain which at first may be general or referred to almost any part of the abdomen, often it is colicky. Within forty-eight hours the pain becomes localized in the right iliac region, and may be continuous or paroxysmal. There are leukocytosis, fever (100°–103° F.), and often frequent micturition, albumin and indican in the urine. *Examination:* A coated tongue. The right thigh is often slightly flexed. On palpation, rigidity of the right rectus; on deep pressure, marked tenderness sharply localized at some point, usually at McBurney's point, between the navel and right superior iliac spine just lateral to the outer border of the right rectus.

Course.—(1) *Recovery:* Most cases after three or four *days* gradually get well and are convalescent in a week or ten days. They may have recurrences at longer or shorter intervals. (2) *Abscess formation:* The symptoms persist, and in four or five days the abscess forms a tumor in the right iliac fossa, visible, firm, circumscribed, tender, and often dull on percussion. This may persist, or rupture into the general peritoneal cavity, a hollow viscus, a blood-vessel, or it may result in pylephlebitis. The temperature is variable, the patient becomes septic. (3) *General peritonitis* follows abscess or perforation of the appendix without adhesions. It may come on at any time, even within a few hours of the onset. There may be sudden sharp pain in the right iliac region, followed by more general abdominal pain, vomiting is frequent, the pulse is rapid (120 or more) and weaker. The symptoms of general peritonitis develop and death follows. (4) *Recurrent or chronic cases:* At intervals of weeks or months there are acute attacks. Finally these may cease spontaneously. Between attacks the patient may be quite well or his general health may suffer.

Prognosis.—A large majority get well without treatment, but there is no certainty of recovery in a given attack.

Treatment should be learned from books on surgery.

QUESTION.

Give the etiology, the varieties, with their lesions and symptoms, and the treatment of appendicitis.

INTESTINAL OBSTRUCTION.

(1) The most common cause is **strangulation.** The gut is kinked or constricted by adhesions from peritonitis following operation; by Meckel's diverticulum; by the appendix; in herniæ into peritoneal pouches, slits in the omentum or mesentery. It is more common in males and in young adult life and in the lower abdomen, especially in the right iliac fossa.

(2) **Intussusception.**—Usually in children. There is an invagination or telescoping of the gut into the gut below. The innermost or *entering layer* is called *intussusceptum;* the middle or *returning layer* is inside out; the outer or *receiving layer* is called *intussuscipiens.* The mesentery is compressed, the gut forms a long cylindrical tumor, and is

congested and swollen. After two or three days firm fibrinous adhesions bind the layers together. The gut may slough. *Mechanism:* The intussuscipiens is supposed to pull itself up and along the inner parts by its longitudinal muscle.

(3) **Volvulus.**—A loop of gut is twisted on the axis of the loop, the mesentery being unusually long; or a piece of gut may be wrapped around another piece of gut; or the gut may be in the shape of a knot. These usually occur in the sigmoid flexure or cecal regin, and are more common in males past thirty.

(4) **Strictures.**—(*a*) Cicatricial from scars of healed ulcers, tubercular, syphilitic, dysenteric, or possibly typhoid. (*b*) New growths; usually cancer of annular shape, rarely benign tumors. (*c*) Pressure from a tumor outside of the gut or even from part of the gut filled with feces. (*d*) Congenital cases are seen very rarely.

(5) **Abnormal contents,** such as impacted feces, sometimes in young children, but usually in the aged; gall-stones that ulcerate into the gut and lodge in the duodenum or ileocecal region; enteroliths (rare); swallowed objects; round worms; magnesium or bismuth masses.

With obstruction, the gut above is distended sometimes to an extreme degree. That below is empty and collapsed. Near the obstruction there are inflammation, local peritonitis, and sometimes necrosis and gangrene.

The **symptoms** may be acute (strangulation, intussusception, volvulus, impacted foreign body or gall-stone) or chronic (fecal impaction, simple or malignant stricture, traction, pressure by outside tumor).

Acute.—There is a sudden onset of abdominal pain, at first colicky, later constant and intense, and perhaps referred to the site of occlusion; repeated vomiting, of stomach contents, then biliary matter, and after one or two days of dark stuff with a fecal odor; constipation usually absolute, although the bowel may empty itself of what it already contains. The abdomen becomes distended, often to an extreme degree, tympanitic, very tender. General symptoms: Cold, clammy skin, pale, pinched, anxious face, dry tongue, extreme thirst, rapid feeble pulse, leukocytosis, shallow breathing, no fever; *marked* prostration. The urine is scanty, concentrated, and contains much indican. The patients die of exhaustion or in coma in from three to six days.

Chronic.—There is irregularly increasing constipation. The stools contain scybala or are slender or flat. There may be attacks of diarrhea or of colic. Vomiting and distention are late symptoms. A fecal tumor may form which can be permanently indented by the finger. Loss of flesh and strength, and anemia may be slight or marked. Chronic obstruction often terminates in an acute or subacute attack that lasts a week or two.

Differential points: (1) *Site:* (*a*) The higher the obstruction the more severe the vomiting, and the sooner it becomes fecal; distention is more localized; the urine more apt to be suppressed. (*b*) Ileum: distention is in the middle of the abdomen, indicanuria very marked. (*c*) Colon: distention is more general and extreme, vomiting later and less; less indicanuria. (2) *The cause*: Age, previous history, and acuteness of attack are suggestive data. (*a*) In *intussusception* mucus and blood are passed, and there are tenesmus and the sausage tumor. The end of the intussuscepted gut may be felt by rectum. It may be subacute, may not be fatal, and the sloughed part of the gut may be passed. (*b*) In *fecal impaction* the fecal tumor is present and feces may be felt by rectal examination. (*c*) Benign strictures develop slowly. (*d*) Cancer gives its general symptoms.

Prognosis.—If untreated an acute attack is fatal (except some intussusceptions that are spontaneously reduced or get well after sloughing).

Treatment.—Chiefly surgical. For intussusception, during the first two days large enemata may be given. For fecal impaction frequent large enemata, especially of sweet oil, are given, and the rectum is emptied by the finger or a spoon. For vomiting, lavage is the best treatment. Purgatives and food by mouth should not be given. Morphin is often needed.

QUESTIONS.

Give the causes and varieties of intestinal obstruction.
Give the symptoms of acute and chronic intestinal obstruction.
How may the cause of the obstruction be diagnosed?
Give the prognosis and treatment.

HABITUAL CONSTIPATION.

Etiology.—*General causes:* (*a*) Often hereditary, more in brunettes. (*b*) Sedentary habits and neglect of defecation. (*c*) Diseases such as anemia, neurasthenia, hysteria, insanity, some organic nervous diseases, chronic liver or stomach disease. (*d*) The habit of taking purgatives. (*e*) A diet with too much or too little residue; too little water or hard waters. (*f*) Prolonged diuresis or sweating.

Local Causes.—(*a*) Weak abdominal muscles, obesity or repeated pregnancy. (*b*) Atony of the colon from colitis. (*c*) Contracted colon after a colitis, or in protracted hysteria or neurasthenia, especially with uterine disease, and in old persons. (*d*) Pressure from outside by tumors, etc., or narrowing of the lumen by foreign bodies or stricture. (*e*) Lead-poisoning. The cause resolves itself into defective expulsive force or inadequate stimulation of the gut.

Symptoms.—Health may be perfect in other respects or there may be a coated tongue, anorexia, a sense of fulness in the abdomen and pelvis, flatulence, palpitation, flushing, malaise, neuralgia of the sacral or visceral nerves, or menstrual disturbances. The colon when full of feces may form a palpable tumor. There may be acute attacks of nausea, vomiting, diarrhea, or of fever. It may result in piles, ulcers of the colon, or formation of scybala or enteroliths in pouches of the colon.

Treatment.—Form a correct habit of defecating at the same hour every day. Support a flabby abdomen by a proper bandage. Use abdominal massage, hydrotherapy, exercise of the abdominal muscles, and horseback-riding. The drinking of much water. More green vegetables and fruit; the correction of any faulty diet. Beside these a suppository of glycerin or, in babies, of gluten; enemata of oil or water; or drugs, such as cascara, aloin, strychnin, belladonna, colocynth, podophyllum, etc., may be needed as a last resort, or as a temporary measure.

QUESTION.

Give the causes and treatment of habitual constipation.

VISCEROPTOSIS (Glenard's Disease, Enteroptosis, Splanchnoptosis).

Etiology.—The causes are repeated pregnancy, recurrent ascites, anemia, tight lacing or wasting diseases. It is more common in women.

One or more of the viscera are abnormally low and movable. Many cases have no subjective symptoms. Girls often have neurasthenic symptoms. The abdominal symptoms, when present, may be indigestion, flatulence, constipation, dragging pain and weakness of the back, especially on exertion.

Physical examination: The stomach (*gastroptosis*) may hang nearly vertically from the cardia; its outlines after inflation (or without it in a person with a flabby abdomen) can be made out by inspection and percussion. *Both* curvatures are low, the greater being below the navel; the vertical axis is long, the *transverse axis is short;* there may be no tympany in the upper part of the normal stomach area. *Enteroptosis* is the sagging of the small intestines; its signs are indefinite. In *coloptosis* the transverse colon may reach the pelvis and is V-shaped; its outlines can be made out after inflation with gas. The liver (*hepatoptosis*) is felt below the ribs, but can be pushed into place. The spleen (*splenoptosis*) may be felt below the ribs and is freely movable. *Movable kidney,* usually on the right side, is common. Any or all of these changes may be present. Sometimes there is kinking with obstruction at the pylorus or the flexures of the colon.

Treatment.—A broad band, supporting the whole anterior wall of the abdomen often gives great relief. Sometimes the liver, spleen, or kidney should be sutured in place. The neurasthenia, anemia, indigestion, and constipation must be treated.

QUESTIONS.

Give the etiology, the symptoms, and treatment of Glenard's disease.

Give the differential diagnosis between gastroptosis and gastrectasis, and between hepatoptosis and an enlarged liver.

NEUROSES OF THE INTESTINES.

MUCOUS COLITIS (Mucous Colic, Membranous Enteritis).

Etiology.—It is more common in adult neurotic women, and may follow irritation of the rectum from riding, fibroids, scybala, etc.

Symptoms.—Habitual constipation is usual. There are attacks of colic and tenesmus, often with a tender area to the left of the navel. In these attacks the patient usually but not always passes large shreds of rather opaque mucus which sometimes form cylindric casts of the gut. *Microscopically,* they contain mucus, with opaque granules, columnar epithelium, leukocytes, cholesterin, and rarely blood. There may be several such stools in an attack. The attack lasts with exacerbations and remissions for from two to ten days. Attacks recur at varying intervals for years. The symptoms of hysteria or nervous depression are usually present.

Treatment.—A purge, irrigation of the colon, rest, and a very restricted diet.

ENTERALGIA (Intestinal Colic).

Enteralgia occurs in many intestinal diseases, but sometimes as a neurosis; from irritants such as food, drugs, chronic plumbism, purgatives, scybala, or worms; with cold, gout, disease of pelvic organs (male or female), emotions or shock.

Symptoms.—Suddenly, or after a few hours of flatulence, there are severe and frequent paroxysms of pain in different parts of the abdomen. They last for a few minutes, and are usually relieved by steady pressure. The patient lies with thighs flexed and is often much prostrated. Constipation or diarrhea may be present. Fever is absent.

Treatment.—Fomentations to abdomen; a purge or enema; sometimes morphin for the pain.

QUESTIONS.

What is mucous colitis?
Give its symptoms.
Give the causes and symptoms of enteralgia.

DISEASES OF THE LIVER.

JAUNDICE.

Etiology.—(1) Obstruction of the ducts. (*a*) The common duct may be closed by swelling of the mucous membrane of the duodenum, or of the duct itself; by a plug of mucus, a gall-stone, parasites such as round worms or flukes, and possibly by food remnants or seeds; by contraction of a scar after ulceration usually due to gall-stones; by cancer of the duct or duodenum; by pressure from tumors of pancreas, stomach, duodenum, colon, liver, omentum, or kidney, or by movable kidney; by a uterus enlarged by pregnancy or tumors, or ovarian tumors; by peritoneal exudates or bands of adhesions; by large nodes in the hilum of the liver; by abdominal aneurysm. (*b*) Obstruction of the ducts within the liver, sometimes of the smallest radicles, is found in many liver diseases.

(2) Toxemic jaundice (hematogenous jaundice) occurs in infectious diseases, such as yellow fever, typhoid, malaria, pyemia, and pneumonia. It occurs in Weil's disease, acute yellow atrophy, and in the newborn. In severe anemias, especially pernicious anemia. With poisoning by phosphorus, arsenic, mercury, chloroform, ether, and snake-venom.

(3) Depressing emotion or shocks may cause jaundice in some way.

Pathology.—In obstruction, the bile is formed but is reabsorbed from the liver. The ducts behind the obstruction are dilated; the common duct may be ½ to 1 inch in diameter, the smaller ducts may have cyst-like dilatations. In long-standing cases there may be new connective tissue in the walls. The obstruction may be confined to the smaller ducts and be caused by acute catarrh or compression; this is seen in some cases called toxemic (phosphorus-poisoning). With marked blood destruction, pernicious anemia, and some infections, there may be no obstruction, but it is supposed that the blood-pigment, transformed to bile-pigment, is in too great excess to be properly excreted, and is reabsorbed by the blood and lymph. The blood and all the tissues contain bile and are stained by it (except nervous tissue). In protracted or intense cases the bile-pigment is precipitated in epithelial cells as granules, and the staining lasts until these cells are desquamated. Bile is present in the urine and sweat, and sometimes in other secretions also.

Symptoms.—*The Ordinary Cases.*—The skin is yellow, dark bronze, or greenish-yellow. There is often very troublesome pruritus, sometimes urticaria, lichen, or boils, rarely xanthelasma. The sweat stains yellow. The visible mucous membranes are yellow, and this is seen most easily on the sclerotics. There are a coated tongue, a foul breath, a bitter taste in the mouth, anorexia, often nausea and vomiting, constipation, and flatulence. With obstruction the stools are clay-colored, greasy, contain much free fat, but no bile, and are very foul smelling. With incomplete obstruction or none, the stools contain bile, and may be very dark. There may be attacks of diarrhea from fermentation. The pulse is slow, sometimes only 30 per minute; the respiration slow; the temperature normal or subnormal, but fever from another cause may be present. Nervous symptoms: Headache, vertigo, irritability, insomnia, mental and physical lassitude, possibly xanthopsia. The urine is darker, yellow, greenish, or brown; the froth is yellow and persistent; the specific gravity is high, a trace of albumin and casts are common. The casts and cells in the urine are stained yellow. The urine stains white paper yellow and it gives the bile-pigment reaction with Gmelin's and other tests.

In the *mildest cases* there is merely yellow staining of the conjunctivæ, skin, and urine without constitutional symptoms. In the *severer cases* there is a tendency to bleeding in the skin, mucous membranes, or kidney; the coagulation-time of the blood is much lengthened. There may be leucin and tyrosin in the urine. *Cholemia* may develop, resembling the typhoid state, with a rapid feeble pulse, restlessness, delirium, twitchings or convulsions, stupor, coma, and finally death after a few days.

QUESTIONS.

Give the causes of jaundice.

Give the symptoms of the ordinary case, a very mild case, and the severer cases.

What is cholemia?

ICTERUS NEONATORUM.

(1) *Mild cases* begin on the second or third day. The child is jaundiced, and has bile in the urine. Sometimes the stools are clay colored after the meconium disappears. The general condition is but little affected. The jaundice lasts a few days or a week and clears up perfectly. (2) *Severer cases* may be due to congenital atresia or stenosis of the common or hepatic duct, syphilitic hepatitis, or septic infection of the umbilical vein. This is fatal usually within a few days. (3) Jaundice is a symptom of the hemorrhagic diseases of the newborn.

ACUTE YELLOW ATROPHY OF THE LIVER.

Etiology.—More common in women from twenty to thirty, especially in pregnancy; but it may occur at any age in either sex. Often the cases occur in groups. It is possible with typhoid, diphtheria, enteritis, syphilis, after fright or other strong emotion, or acute alcoholism. Very similar symptoms may develop in hypertrophic cirrhosis or in acute phosphorus-poisoning. It is supposed to be either an infectious disease or else an auto-intoxication.

Lesions.—At first the liver may perhaps be enlarged, but at autopsy it is very small, weighs 20 to 30 ounces, is wrinkled and flabby. On section the surface is yellowish or mottled with red areas. The gall-ducts and bladder are patent but empty. Microscopically, the yellow areas show extreme fatty degeneration of the liver-cells. In the redder areas the cells may have disappeared, leaving a mass of granular débris, leucin spheres, tyrosin needles, granules of pigment, and cholesterin crystals. In places there may be proliferation of the connective-tissue cells and sometimes of liver-cells. The spleen, portal, and retroperitoneal lymph-nodes are large and soft. There is fatty degeneration of the cells of the kidneys, pancreas, mucous membrane of the stomach and intestine, and of the heart and other muscles; and the lesions of jaundice.

Symptoms.—There may be a prodromal stage with gastro-intestinal symptoms and sometimes a mild jaundice. This may last a week or two, and then the patient becomes rapidly ill with the intense symptoms of jaundice, bleeding into the skin, mucous membranes, kidneys; leucin spheres and tyrosin needles and very little urea in the urine; and cholemia. Fever is absent until shortly before death. There are the physical signs of a rapidly diminishing liver, the dulness receding daily until it may be almost wholly lost. The patient dies within a week. A few instances of recovery have been recorded.

Treatment is unavailing.

The *other forms of toxemic jaundice* are usually mild and bile is found in the stools.

QUESTION.

Give the etiology, lesions, symptoms, and prognosis of acute yellow atrophy of the liver.

DISEASES OF THE BILE-DUCTS AND GALL-BLADDER.

ACUTE CATARRHAL CHOLANGITIS (Acute Catarrhal Jaundice).

Etiology.—Probably always an extension of inflammation from the duodenum. It is most common in young persons. It may follow acute indigestion, exposure to cold, errors in diet, portal obstruction, chronic Bright's or heart disease or malaria, pneumonia, typhoid or other infectious diseases. Possibly the emotional jaundice belongs here. Sometimes it occurs in small epidemics.

Lesions.—The orifice of the common duct is occluded by swelling of the duodenal mucous membrane, or that of the lower end of the duct itself, or by a plug of mucus. There are the lesions of the duodenal catarrh and of jaundice.

Symptoms.—The attack begins with digestive disturbance due to duodenitis, after a few days jaundice and its symptoms (p. 178) appear. It lasts from one to six or eight weeks. In recovery the general symptoms disappear first, the discoloration persists, sometimes for weeks. First the color returns to the stools, then it disappears from the skin and sclerotics, and finally from the urine. During the first part of the attack there may be tenderness over the liver, and its edge may be felt an inch below the ribs in the right nipple-line. Sometimes the gall-bladder may be felt to be enlarged. Percussion may make out enlarged areas of dulness over these organs. There are *mild cases* with no general symptoms, and only the discoloration of skin, eyes, and urine, and the colorless stools. They may last only a few days. There are *severer cases* in which the patient is quite ill with severe gastroduodenitis, fever, and prostration. When this occurs in a person with a cirrhotic or fatty liver it may be fatal.

Treatment.—The duodenitis and gastritis need treatment. For the jaundice the diet should be light and free from fats. At first only liquids may be given, later light starchy foods or lean meat is added. Much water, especially alkaline waters, should be taken. The bowels should be moved at first with calomel, and afterward with salines taken daily. Daily enemas of two quarts of cold water are given. Most cases do not stay in bed. For the itching, lotions of borax or soda (5 per cent. in water) or of menthol (2 per cent. in alcohol) are used. Other symptoms may need treatment.

QUESTION.

Give the etiology, lesions, symptoms, and treatment of acute catarrhal jaundice.

CHRONIC CATARRHAL CHOLANGITIS.

Etiology.—Protracted obstruction of the common or hepatic ducts, due usually to impacted stone, but also to cancer, stricture, or external pressure. Possibly it may follow acute cholangitis.

(1) *With Complete Obstruction of the Common Duct.*—Above the obstruction the ducts are much dilated, the common duct admitting a finger. The bladder also is dilated. The contents are mucus, free from bile and often sterile. The mucous membrane is usually smooth. The symptoms are those of persistent and marked jaundice without bile in the stools and with a tendency to bleeding.

(2) *With Incomplete Obstruction of the Common Duct.*—The usual cause is an impacted stone, but it may be due to external pressure. The ducts are less dilated and the bladder not dilated; it is usually contracted. The contents are bile and mucus. The most characteristic symptoms are *intermittent hepatic fever of Charcot.* This is probably due to an infection. There are paroxysms of chill, fever (103°–105° F.), and often sweats. They recur at regular or irregular intervals for weeks, sometimes resembling intermittent malaria. Often with the paroxysm there is pain like that of biliary colic, with colorless stools. After the attack the jaundice deepens for a few days. The gall-bladder is not dilated; there is no suppuration; the general health may not be affected.

Treatment is that of the causative factor and of the jaundice, and biliary colic.

Courvoisier's Rule.—With impacted stone in the common duct the gall-bladder is seldom dilated; with cancer and many other obstructions it is dilated.

QUESTIONS.

Give the etiology and symptoms of complete and of incomplete obstruction of the common duct.

What is intermittent hepatic fever?

What is Courvoisier's rule?

SUPPURATIVE AND ULCERATIVE CHOLANGITIS AND CHOLECYSTITIS.

Etiology.—Colon bacillus, typhoid bacillus, streptococci and staphylococci of suppuration, or the pneumococcus are found. Infection and the growth of bacteria are favored by gall-stones, also by cancer of the duct or by foreign bodies; sometimes by suppurative pylephlebitis or liver abscess. Typhoid and pneumonia are important causes.

Lesions.—There is suppurative inflammation of all of the gall-ducts and the bladder. The ducts are dilated, and those within the liver may form abscesses. The bladder may be dilated, and usually contains gall-stones, pus, and bile. The ducts contain pus and bile; their walls are thickened, and often adherent to contiguous structures. There may be ulceration and perforation of the colon, duodenum, abdominal wall or elsewhere, but usually into the peritoneum with local abscess or general peritonitis.

Symptoms.—There is an abrupt or more gradual onset of septicemia or pyemia with all of their symptoms; also pain and tenderness over the region of the liver and gall-bladder and the epigastrium; jaundice of varying intensity with little or no bile in the stools. *Physical Examination:* The physical signs are those of a large tender liver, its edge may be felt at the level of the navel, and of an enlarged gall-bladder (p. 185), and often those of local peritonitis. The patient dies of sepsis in a few days or weeks, or of general peritonitis.

Treatment is purely surgical. If early enough, an operation may perhaps be followed by recovery.

ACUTE INFECTIOUS CHOLECYSTITIS.

Etiology.—Some cases seem to be primary infections without obvious cause, but usually the etiology is that of suppurative cholangitis and cholecystitis.

Lesions.—(1) **Catarrhal Form.**—The cystic duct is usually closed, the gall-bladder is dilated, shows catarrh of its mucous membrane, and contains mucus, bile, and usually gall-stones.

(2) **Suppurative or Phlegmonous Form.**—The cystic duct is usually closed, the bladder dilated and distended with pus, mucus, often foul-smelling, and sometimes blood. Gall-stones are common. The wall is swollen and edematous. It may be ulcerated or phlegmonous, with sloughing or rarely with gangrene. There usually are peritoneal adhesions. There may be perforation.

Symptoms.—*Suppurative or Phlegmonous Cholecystitis.*—There are an acute onset of intense sepsis with severe colicky abdominal pain referred to the hypochondrium, below it, or to the epigastrium; nausea, vomiting; tympanites; constipation (sometimes absolute); the *physical signs* of a large tender liver that may reach the navel, and of a large tender gall-bladder. Jaundice is usually absent. In a few days the patient dies of sepsis, or of general peritonitis from perforation. The diagnosis from appendicitis or intestinal obstruction may be made at operation or autopsy. After early operation the patient may recover.

Catarrhal Form.—The symptoms are similar but less intense, and recovery is common.

Treatment is purely surgical.

EMPYEMA OF THE GALL-BLADDER.

Etiology.—Obstruction of the cystic duct, with stones, or abscess of liver, and the same bacteria as above.

Lesions.—The cystic duct is occluded, the gall-bladder much distended and full of pus. It usually contains gall-stones. The wall is thickened. The tumor hangs down from the liver like a pouch, and may contain a pint or quart of liquid. Adhesions are often present.

Symptoms.—This is really a less acute form of the preceding condition. With an acute or more gradual onset the same symptoms in milder form are present. The patient may not be bedridden for a week or more. The condition may last several months with chronic sepsis and emaciation. There may be rupture, externally forming a sinus that discharges pus, often free from bile, with gall-stones; or into the intestine with formation of a fistula; sometimes into the pleura or lung; or into the general peritoneal cavity, or it may form a local peritoneal abscess. The patients die of chronic sepsis, peritonitis, or of pneumonia, especially of the right lower lobe. With operation they may recover.

The **treatment** is purely surgical.

QUESTION.

Give the etiology, lesions, symptoms, prognosis, and treatment of suppurative cholangitis, of acute infectious cholecystitis, of empyema of the gall-bladder.

CHRONIC OR RECURRENT CATARRHAL CHOLECYSTITIS.

There is a group of cases with repeated attacks like biliary colic without jaundice. The gall-bladder is distended with mucus and contains stones.

DROPSY OR HYDROPS OF THE GALL-BLADDER.

Etiology.—Occlusion of the cystic duct, usually from impacted stone, or sometimes following ulceration due to stone, with adhesions or cicatricial contraction. There is no marked infection.

Lesions.—This group gives the largest gall-bladders. The cystic duct is closed. The gall-bladder dilated, sometimes enormously so. It contains clear water and mucus and sometimes gall-stones. Several quarts of liquid have been found in it. The anterior edge of the liver is prolonged downward like a tongue (Riedel's lobe).

Symptoms.—Simply those of tumor of the gall-bladder. When marked there may be a feeling of tension and dragging in the right side of the abdomen with some indigestion and at times palpitation and dyspnea. *Physical signs:* Inspection. With thin abdominal walls there is often a visible rounded tumor on the right side of the abdomen, in extreme cases reaching the pelvis or nearly filling the abdomen. If not too large or adherent, it is pyriform in shape and moves with the liver in breathing. On palpation the tumor feels tense, is movable, and can be traced to the edge of the liver. Here the flat hand usually feels a groove where it joins the liver. Riedel's lobe and the notch in the liver can be felt. Occasionally gall-stone crepitus can be felt. On percussion the note is flat over the tumor and this is continuous with the liver dulness above. Occasionally intestine gets in front of the tumor and modifies this sign. An aspirating-needle would find clear watery mucus, and it might give a decisive feel as it rubs against a stone. The needle, however, is seldom used.

The **treatment** is surgical.

QUESTION.

Give the etiology, the lesions, the physical signs, and the treatment of dropsy of the gall-bladder.

CARCINOMA OF THE GALL-BLADDER.

(1) **Primary Cancer.**—Usually in women over forty, and gall-stones are almost always present. The primary growths usually begin at the fundus of the bladder, and may form

large tumors that are nodular, tender, painful, and very hard. The pain may be constant or paroxysmal, like biliary colic. Jaundice is not rare. Secondary tumors appear in the liver and elsewhere. There are all the general symptoms of cancer and death within a year.

(2) **Secondary tumors** of the gall-bladder may begin in any part of it and gall-stones are often absent.

CARCINOMA OF THE COMMON BILE-DUCT.

Primary cancer is usually near the duodenum and causes very persistent jaundice with no bile in the stools and with dilatation of the gall-bladder (Courvoisier's rule). It is unusual to feel the tumor. There are the general symptoms of cancer.

Treatment.—Tumors of the gall-bladder may be successfully removed by an early operation. Removal of tumors of the duct is less successful.

QUESTION.

Give the etiology and symptoms of carcinoma of the bile-passages.

STENOSIS OF THE COMMON DUCT.

The **etiology** has been given with the causes of jaundice (p. 177). Impacted stone is the most common cause.

(1) **With Complete Obstruction.**—The liver may be large, but in time it is often reduced in size. The gall-bladder is much dilated and contains colorless liquid and perhaps gall-stones. No bile enters the duodenum. The patient may live a year or two with intense jaundice.

(2) **Incomplete Obstruction.**—This is usually due to impacted stone. The gall-bladder is not dilated (Courvoisier's rule). The *symptoms* are those of variable or intermittent jaundice and sometimes hepatic intermittent fever.

CHOLELITHIASIS (Gall-stones).

Etiology.—Much more common in women over thirty *years; especially* in those who have borne children. It is predisposed to by a sedentary life, tight lacing, splanchnoptosis, a diet deficient in proteids and water and with excess of starches, by typhoid fever, and other infections, catarrh of the gall-bladder, and stasis of bile.

The stones may be single or multiple, even many hundreds. In size they range from biliary sand to a stone four inches long or even larger. The largest stones are single and form rough molds of the gall-bladder. Ordinary stones (¼–¾ inch in diameter) are usually ovoid or spheroidal, or if multiple, polyhedral and facetted. The *surface* is usually smooth, but sometimes nodular (mulberry stones). The *color* ranges from white to almost black; usually it is yellow. In *consistence* some are waxy, others stony with crystalline fracture, and some very friable. *Composition:* Cholesterin forms the greater part of most stones. Other constituents are lime carbonate, bile-pigment and salts, mucus, and a *nucleus* of epithelial cells, bacteria, dried mucus, precipitated bile-pigment or rarely a foreign body. Around the nucleus the cholesterin is laminated, often radially striated, and of waxy consistence and greasy feel. Rarely it is pure and white, but usually is mixed with bile-pigment and is yellow. The laminæ are cemented by mucus and other substances. On the surface there is often some lime salts.

Formation.—(*a*) Bacteria lodge in the gall-bladder and set up a mild catarrh. The catarrhal mucous membrane secretes more cholesterin and lime salts than normally. (*b*) Stagnant bile contains less bile salts, and this lessens its solvent power for bile-pigments and cholesterin and they are precipitated. When these factors coexist the stones are formed around a nucleus. Associated with gall-stones there may be almost any of the various conditions previously described.

Symptoms.—(1) **Biliary or Hepatic Colic.**—The typical case is due to the passage of a stone of moderate size from the gall-bladder, through the cystic and common ducts into the intestine. There is a very abrupt onset of pain, intense and paroxysmal, referred to the region of the gall-bladder and radiating over the abdomen, and upward to the right scapula and shoulder. With this are chills, fever (101–103° F.), nausea, vomiting, more or less collapse, with cold sweats, rapid, feeble heart and pulse, shallow breathing, and perhaps syncope. *Examination:* There are tenderness over the gall-bladder (between the tip of the ninth rib cartilage and the navel), and a rigid right rectus. After a few hours or a day or two the liver and gall-bladder may be large and tender and the spleen large. The urine may contain some albumin and red blood-cells. The attack lasts from a few hours to a few days, and is followed by soreness over the gall-bladder. The stone may be found in the stools if they are carefully strained. In the longer attacks there are marked remissions of the pain, and if the stone is delayed in the common duct jaundice appears on the second or third day. It lasts longer than the other symptoms.

(2) **Stone in the Gall-bladder.**—(*a*) No symptoms have been present in most cases found at autopsy. The gall-bladder is often contracted around the stones. Other cases have (*b*) repeated attacks like biliary colic, but without jaundice or dilated gall-bladder. This may seriously affect the general health. Or (*c*) various inflammations of the gall-bladder, perforation, or peritonitis.

(3) **Stone in the Cystic Duct.**—(*a*) There is biliary colic when the stone enters the duct. If the stone is impacted, the colic is repeated at intervals and the gall-bladder is dilated and tender at every attack, but there is no jaundice. (*b*) Other results are hydrops, empyema, or other inflammations of the gall-bladder, a small atrophied or calcified gall-bladder, or ulceration of the stone through the wall of the duct.

(4) **Stone Impacted in the Common Duct.**—(*a*) Biliary colic when it enters the duct. (*b*) Later attacks of colic with jaundice at varying intervals for months. (*c*) Infective cholangitis with hepatic intermittent fever (p. 182). Obstruction is rarely complete. (*d*) Suppurative cholangitis may develop. (*e*) The stone may ulcerate through the wall of the duct into the duodenum or elsewhere.

(5) **Gall-stones in the intestine** are usually passed without trouble, but may lodge, grow by deposits of lime salts, and form large enteroliths, which may cause acute or chronic intestinal obstruction.

(6) **Biliary fistulæ** usually form from the gall-bladder, rarely from the ducts. They open into the duodenum; colon; externally in the epigastric or hypochondriac region;

into the peritoneum, forming a peritoneal abscess or general peritonitis; rarely elsewhere.

Treatment.—(1) *Biliary colic:* A hot bath, hot epigastric fomentations, hot alkaline drinks, saline laxatives, large enemas of hot water, and often morphin hypodermically are used. (2) *To prevent further formation of gall-stones:* Courses of treatment at alkaline springs may help. Also, a diet including much water and but little starches and sugars. Much exercise is advisable. (3) *Surgical treatment* is indicated with repeated attacks of biliary colic, a large gall-bladder from any cause with or without pain, hepatic intermittent fever, suppurative or phlegmonous inflammation of the bile-passages, perforation, peritonitis, and biliary fistulæ.

QUESTIONS.

Give the etiology and pathology of gall-stones.

Give the symptoms of biliary colic.

Give the symptoms of stone in the gall-bladder, of stone in the cystic duct, of stone impacted in the common duct, and of gall-stones in the intestine.

Discuss biliary fistulæ.

Give the treatment of cholelithiasis.

THE BLOOD-VESSELS OF THE LIVER.

PORTAL OBSTRUCTION.

Etiology.—(1) **In the smaller branches within the liver** obstruction may be due to atrophic or fatty cirrhosis, carcinoma, or syphilis of the liver. (2) **Obstruction to the trunk or large branches** may be due to (*a*) thrombosis or embolism of the portal vein, to (*b*) pressure from without by tumors or large lymph-nodes at the hilum of the liver, and chronic productive peritonitis.

Collateral and Compensatory Circulation.—(1) The accessory portal circulation of Sappey includes the anastomoses (*a*) of veins on the surface of the liver, with phrenic branches and the veins of the gastro-epiploic omentum; and (*b*) of a para-umbilical vein in the round ligament with mammary and epigastric branches of the abdominal wall. (2) There are anastomoses of the retroperitoneal veins, with the renal veins. (3) Of the gastric with the esophageal veins at the cardia. (4) And of the hemorrhoidal veins in the hemorrhoidal plexus. All of these veins beyond the point of obstruction become dilated and often varicose.

Symptoms.—With a slow onset of obstruction the collateral circulation may be adequate and no symptoms develop. In time it is apt to fail, then there may be epistaxis, rupture of the esophageal veins with regurgitation or vomiting of blood; bleeding into the stomach and intestine, which may be profuse and repeated; bleeding piles; the symptoms of chronic gastritis and enteritis; persistent ascites; perhaps edema of the lower limbs; the physical signs of a large spleen; dilated veins on the anterior abdominal wall which may form the caput medusæ (a ring of varicose veins around the navel, with radiating serpentine branches). With gradual obstruction the symptoms develop gradually and are usually quite persistent. With sudden obstruction the symptoms develop rapidly, but later, in some cases, they may abate as the collateral circulation becomes more efficient.

The **treatment** is that of the causes and the various symptoms.

QUESTION.

Give the etiology and the symptoms of portal obstruction.

THROMBOSIS AND EMBOLISM OF THE PORTAL VIENS.

Thrombosis is found in syphilis, cancer, and cirrhosis of the liver; with pressure on the portal vein from tumors, gallstones, or productive peritonitis; it may occur in cachectic conditions, or with infections of the abdominal viscera. Embolism may follow appendicitis or intestinal or gastric ulcers. It is often septic.

Lesions.—Thrombi fill a branch or the main trunk of the vein; they are red or white. Rarely the clot becomes replaced by a mass of fibrous tissue. With embolism infarction is rare, the collateral circulation being generous. With infected clots abscesses form. Symptoms often are not marked, but when the main branch is affected there are sudden and severe symptoms of portal obstruction. Occasionally the collateral circulation becomes more efficient and the symptoms abate somewhat. With infected clots abscesses and suppurative pylephlebitis may follow.

The **prognosis** is that of the cause, but usually is bad.

Treatment is that of the cause and of the portal obstruction.

SUPPURATIVE PYLEPHLEBITIS.

Etiology.—Infection comes from the tributaries of the portal vein, usually from the appendix, also from ulcers of the stomach, intestine, or any other focus.

Lesions.—Besides the primary lesions there is an enlarged liver with a mottled surface. On section the portal branches may be full of pus, and abscesses are scattered throughout the liver. They communicate with the veins, and may contain bile-stained pus. The spleen is large and soft. Later the lesions of pyemia develop.

Symptoms.—Besides those of the primary disease there are pain and tenderness over the liver and the signs of an enlarged liver and spleen. Jaundice is not rare, but the stools contain bile. There are the symptoms of septicemia and pyemia, and death in one to four weeks.

Treatment is unavailing.

QUESTIONS.

Give the causes, lesions, and symptoms of thrombosis and embolism of the portal veins.

Give the causes, lesions, and symptoms of suppurative pylephlebitis.

CHRONIC CONGESTION OF THE LIVER

(Passive Hyperemia).

Etiology.—(1) Chronic heart disease of the right side or mitral valves. (2) Emphysema or chronic interstitial pneumonia. (3) Less often from pressure on the hepatic vein or inferior vena cava from intrathoracic tumors, or nodes or other tumors at the hilum of the liver. (4) Very rarely from disease or thrombosis of the hepatic veins.

Lesions.—The liver is enlarged, dark red, and firm. It cuts with increased resistance. On section the surface is mottled (nutmeg liver); the center of the lobule is dark red or brown, the periphery is yellowish. The tributaries of the hepatic vein are dilated. *Microscopically:* At the center of the lobule the capillaries are dilated; later the liver cells atrophy and pigment is abundant; finally, new connective tissue may develop. At the periphery of the lobule there may be fatty degeneration of the liver-cells. There is proliferation of the connective tissue between the lobules. In time the contraction of the new connective tissue may result in a small liver.

Symptoms.—Those of the primary condition. Besides these there may be dragging or pain over the liver. *Examination:* The liver edge may be felt as much as four inches below the ribs in the nipple-line; the surface is smooth and somewhat tender; the liver may pulsate. There may be jaundice of varying intensity (usually with some bile in the stools), portal obstruction, and chronic gastritis.

The **prognosis** is that of the primary disease.

The **treatment** is that of the primary disease and symptomatic. Saline purges with little water, or the aspiration of ten to twenty ounces of blood direct from the liver may relieve.

QUESTION.

Give the causes, lesions, and physical signs of chronic congestion of the liver.

CIRRHOSIS.

(1) **Atrophic Cirrhosis.**—*Etiology:* Usually in males in middle life. (*a*) The drinking of alcohol, especially spirits, is the most important factor. (*b*) Sometimes spicy foods, gluttony, and indigestion seem to predispose. (*c*) Occasionally it follows scarlatina, syphilis, rickets, diabetes, gout, cancer, tuberculosis, possibly malaria. (*d*) Passive congestion.

(2) **Fatty Cirrhosis.**—The same etiology, except that it is caused by malt liquors, usually with obesity. "*Alcoholic cirrhosis*" includes these two forms.

Lesions.—**Atrophic Cirrhosis** (hob-nailed liver).—In the early stage the liver is large and occasionally remains so.

Later, usually it is small, two pounds or less, distorted, rough, dimpled, and hard; it cuts with great resistance, and is tough; the capsule is thick. The cut surface is rough and yellowish, projecting areas are seen on a grayish ground. *Microscopically:* Connective-tissue proliferation begins around the portal veins, causing portal obstruction. The connective tissue *surrounds groups of several lobules.* Later this tissue contracts. Liver-cells atrophy, especially at the periphery of the lobule. Bile-capillaries may be increased.

Fatty Cirrhosis.—The liver is enlarged, smooth, not hard, light yellow, cuts with resistance. *Microscopically:* The same connective-tissue changes, with fatty degeneration of the liver-cells. In both there are the lesions of portal obstruction, chronic catarrhal gastritis and enteritis, and often interstitial nephritis, and arteriosclerosis with its heart-changes.

Symptoms.—Often a history of alcoholic gastritis. Liver symptoms are absent while the compensatory portal circulation is adequate. When this fails, there are all the symptoms of portal obstruction (p. 190). Jaundice may be a late symptom, and usually is slight. Fever varies, but usually is present at some time. The urine is heavy, with a sediment of urates, diminished urea, increased indican; albumin is not common. *Physical examination:* Hepatic facies; the face is thin, the eyes sunken and watery, the venules of the cheek distended; the skin is muddy and icteroid. There are the physical signs of portal obstruction. After the ascitic fluid has been removed in atrophic cirrhosis the liver-dulness is normal or reduced, the edge is hard and rough; but early in the disease it may be tender.

In **fatty cirrhosis** the symptoms are the same, but cases without symptoms are more common. *Physical examination:* Even before tapping, the surface of the liver can often be felt, and its edge mapped out, perhaps four inches below the ribs in the nipple-line. After tapping the liver is more easily made out, its surface is hard and smooth.

Toxic Symptoms.—Symptoms resembling cholemia (p. 178), even without jaundice, may develop in either type.

Course.—Marked cirrhosis with no symptoms may be discovered at autopsy. The patient usually dies in from six months to one year after the onset of ascites, from the toxic symptoms, tuberculosis, especially of the peritoneum or lungs, or the associated lesions in the heart, arteries, kidneys, or from intercurrent disease, rapidly recurring ascites, or bleeding. Rarely the symptoms ameliorate or disappear, the patient living for years.

(3) **Hypertrophic Cirrhosis of Hanot.**—*Etiology:* Usually in males below middle life, and not rare in children. *Lesions:* The liver is large, four to six pounds or more, hard, dark greenish yellow, smooth or granular, and cuts with resistance. *Microscopically:* New connective tissue, often rather cellular, surrounds *individual lobules.* The liver-cells are but little degenerated, and may be large and mitotic; new bile-ducts are abundant. The larger ducts are patent; the smaller show productive and catarrhal inflammation. There are the lesions of jaundice, and the spleen is large. *Symptoms:* Jaundice is persistent, but often of moderate degree. The stools contain bile; they may be very dark. There is a tendency to bleeding; there may be icterus gravis and cholemia. *Liver crises* occur, with vomiting, some fever, paroxysmal pain in the liver, followed by more intense jaundice. There may be dragging pain in the liver. *Physical signs:* On inspection a distinct fulness over the liver; *on percussion,* the dulness may extend from the sixth rib to a few inches below the ribs in the nipple-line; *on palpation,* the surface is smooth, hard, not tender, the edge is sharp, hard, and may be below the navel. The spleen is large, hard, and may extend three inches below the ribs. The symptoms of portal obstruction are absent. The fever varies much, but is common with the crises, and may last a week or two. There is a leukocytosis. *Course:* The disease lasts from two to six years, with persistent jaundice, and gradual loss of flesh and strength. They die of icterus gravis, with bleedings from the skin and mucous membrane, of emaciation, or of intercurrent disease.

(4) **Biliary cirrhosis** follows chronic obstruction of the large gall-ducts; it is marked by deep jaundice and a moderately enlarged liver with new connective tissue around the smaller bile-ducts. *Microscopically* it is like hypertrophic cirrhosis. The symptoms are those of intense jaundice with white stools and the symptoms of the causative condition. There are the physical signs of a moderately large liver.

(5) **Syphilitic Cirrhosis.**—The important points are the history and symptoms of syphilis; the most characteristic liver is large, rough, and very nodular, with the symptoms of atrophic cirrhosis. The condition is often curable.

(6) **Capsular cirrhosis (perihepatitis)** is associated with chronic productive peritonitis, perisplenitis, chronic interstitial nephritis, and syphilis, hereditary or acquired. *Lesions:* The liver is very small, rounded, and firm; cuts with extreme resistance near the surface, but is softer inside. The capsule is thick, ¼ inch or more. The inner part is soft, the cells are but slightly degenerated. There are the symptoms of portal obstruction and the associated conditions. The liver when palpable has a rounded edge. The *prognosis* is bad.

Treatment of Cirrhosis.—In alcoholic cirrhosis, eliminate the cause and treat the portal obstruction. In syphilitic cirrhosis give antisyphilitic treatment. Otherwise the treatment consists of hygienic treatment and the treatment of the associated conditions.

QUESTIONS.

Give the etiology, lesions, and symptoms of atrophic cirrhosis of the liver.

Give the etiology, lesions, and symptoms of hypertrophic cirrhosis.

Give the diagnostic points of syphilis of the liver.

What is the treatment of cirrhosis?

ABSCESS OF THE LIVER.

Amebic Abscess (Large, Solitary, or Tropical Abscess).—*Etiology.*—It is due to or associated with the Amœba dysenteriæ, but often streptococci and staphylococci of suppuration, or the colon bacillus are present. It usually follows amebic dysentery, but sometimes occurs without it, is usually in males and in tropical countries.

Lesions.—The affected part of the liver, usually the right lobe near the convex surface, shows a large, rounded swelling which may fluctuate. On section there is a large abscess, the contents are a reddish-brown, thick pus; sometimes creamy or mucoid or bile-stained, and often putrid. It may amount to from 1 to 6 pints or more. It is often sterile. *Microscopically* it contains amebæ, degenerated liver cells, blood pigment, leukocytes, and detritus. The wall is gray, necrotic, contains amebæ, pus cells, necrotic liver tissue, and often bacteria. Surrounding this is a zone of brownish-red liver-tissue and around this is congested liver. In old cases there may be a dense fibrous wall. Sometimes there are two or three large abscesses which may run together. Rarely there are many small abscesses containing amebæ.

Symptoms.—It usually follows dysentery. There are symptoms of sepsis; the *fever* varies extremely; it may be intermittent with paroxysms of chill, fever (103° F. or higher), and sweating. *Pain,* referred to the liver or the back of the right chest or to the right shoulder, varies greatly in acuteness and duration. There may be a dragging sensation in the liver, especially when the patient is erect or on the left side. There are often a slight jaundice, marked leukocytosis, and a progressive loss of flesh and strength. *Physical signs:* On inspection there may be a prominence of the right lower ribs. Liver dulness extends upward to the ninth, eighth, or even seventh right rib behind, and in front nearly to the nipple. Downward, it may reach 2 or 3 inches below the ribs. On palpation there may be deep tenderness, rarely fluctuation, or a peritoneal rub. There may be the signs of a compressed or retracted lung at the right base. The complexion is muddy and slightly jaundiced. Some *chronic cases* have no noticeable symptoms for weeks or months, but periods of exacerbation follow. The abscess may *rupture* into the right pleura, forming an empyema; into the right lung, with pain in the lung, spasms of coughing, the expectoration of pus, often reddish brown, containing amebæ and liver tissue; into the peritoneum or colon or externally.

Duration.—The most acute cases last four to six weeks, the chronic cases may last a few years. If untreated, patients die of rupture or of chronic sepsis.

Treatment.—*Preventive:* Treat all cases of colitis vigorously. *Curative:* Operation is often successful.

Septic and Pyemic Abscesses.—*Etiology.*—Streptococcus and staphylococci of suppuration, the colon bacillus, the typhoid bacillus, or the pneumococcus. They reach the liver (*a*) through the portal vein (see p. 191), suppurative pylephlebitis); (*b*) through the gall-ducts (see Suppurative cholangitis); (*c*) through the hepatic artery from malignant endocarditis, or other pyemia; (*d*) through the hepatic vein, rarely; (*e*) directly through a wound, or by extension from contiguous surfaces. (*f*) Infection may follow contusion of the liver or an injury to the head. (*g*) Rarely, an echinococcus cyst suppurates. *Lesions:* The liver is large, mottled, and usually contains many small abscesses; less often a few or even one large abscess. There are the lesions in the portal vein, the gall-ducts, etc. *Symptoms:* Practically those of suppurative pylephlebitis or suppurative cholangitis. *Treatment:* Large single abscesses may be opened. Other cases are treated like septicemia.

QUESTIONS.

Give the etiology, lesions, and symptoms of amebic abscess of the liver.

Give the etiology, lesions, and symptoms of pyemic abscesses of the liver.

Give the treatment of liver abscesses.

FATTY LIVER.

Etiology.—Without other liver disease *fatty infiltration* may follow excessive eating, alcohol, and obesity. *Fatty degeneration* is most marked in phosphorus-poisoning and acute yellow atrophy; it occurs also in the cachexia of profound anemias, tuberculosis, or cancer.

Lesions.—The ordinary fatty liver is large (perhaps five or six pounds), pale, smooth, soft, cuts easily, leaving the knife greasy; it may float. *Microscopically*, there are granules and drops of fat in the liver cells.

The ordinary cases present no **symptoms** except the physical signs of a large, smooth liver that is not hard or tender.

The **treatment** is that of the causative condition.

AMYLOID DISEASE.

Etiology.—This follows (*a*) chronic suppuration, especially of the bones or joints; (*b*) tuberculosis of the bones or joints; (*c*) syphilis; (*d*) chronic malaria, leukemia, pseudoleukemia, rickets, or cancer.

Lesions.—It involves the liver, spleen, kidneys, often the intestines, and sometimes other organs. The *liver* is large, six or eight pounds, or even more, smooth, pale, firm; the cut surface glistens, becomes mahogany brown when wet with Lugol's solution of iodin. *Microscopically:* The capillary walls are thickened, homogeneous, and give the amyloid reaction; the lumen is narrow. The interlobular veins and the connective tissue are similarly changed in advanced cases. The liver-cells are but little affected.

Symptoms.—Chiefly those of the causative condition, with the physical signs of a large, hard, smooth, painless liver, with a rounded edge that may be below the navel. There is no jaundice or portal obstruction. Associated with this are the large hard spleen, the symptoms of amyloid kidney, and often a persistent diarrhea, with mucous stools.

The **treatment** is that of the causative condition.

CARCINOMA OF THE LIVER.

Etiology.—*Primary cancer* is rare. *Secondary growths* follow tumors of the breast, stomach, cervix uteri, or other organs. They are usually seen after forty, and are more common in males. Heredity and trauma may predispose.

Lesions.—Primary cancer is usually (*a*) a single large tumor; sometimes (*b*) many nodules are scattered throughout the liver; very rarely (*c*) "cancer with cirrhosis." With secondary cancer nodules are scattered throughout the liver. With a single large primary tumor or with secondary tumors the liver may be enormous, even twenty pounds. On section the cancer masses are whitish, and may have a softened center. *Microscopically,* they are medullary carcinoma, adenocarcinoma, or the type of the primary tumor. The liver tissue shows fatty or hyaline degeneration, and some new connective tissue. There may be bleeding or suppuration in the tumor.

Symptoms.—*Pain* usually is present; it may be mild or extreme. *Jaundice* is present, but may be moderate. *Portal obstruction* is present in a few cases. *Fever* is usually present *at some time.* There are leukocytosis, the general symptoms of cancer and those of the primary tumors. *Pysical signs:* Inspection shows a fulness in the hepatic region and below it; on palpation the edge of the liver is felt below the navel, the surface and edge are rough, nodular, hard, and often pitted. The tumor moves with respiration. On percussion, the liver dulness is enlarged both downward and upward. There may be the symptoms and signs of cancer of the peritoneum also. Patients may live from six to fifteen months. They die of exhaustion, of intercurrent disease, or of symptoms like cholemia.

The **treatment** is symptomatic and palliative.

QUESTIONS.

Give the varieties and etiology of fatty liver.
Give the physical signs of the large fatty liver.
Give the etiology, lesions, and symptoms of amyloid liver.
Give the etiology, lesions, and symptoms of carcinoma of the liver.

DISEASES OF THE PANCREAS.

PRELIMINARY CONSIDERATIONS.

With pancreatic disease areas of *necrosis of fat* are found in and around the pancreas, in the omentum, mesentery, and less often elsewhere. These areas look like tallow, and may be microscopic or from a quarter to half an inch in diameter. They are probably due to a fat-splitting ferment derived from the pancreas. This condition is not always fatal.

A common symptom-group in pancreatic disease is the *pancreatic crisis*, marked by an abrupt onset of intense paroxysmal pain referred to the epigastrium, which may radiate to the back or around the trunk. With this are nausea and persistently repeated vomiting. The vomitus may contain bile, and after a time blood. There are flatulence, fulness and tenderness in the epigastric region, rigidity of the recti muscles, constipation, faintness, often collapse with clammy skin, and a rapid, feeble pulse. These crises sometimes are fatal; in other cases the pain may become intermittent or gradually die out.

With *absence of the pancreatic secretion from the intestines* the stools usually contain an excess of fatty acids and of soap. They may look oily and light colored, even when bile is present. They contain an excess of undigested proteids. They are often bulky and very offensive. Rarely a fatty diarrhea is seen.

With most pancreatic diseases there is a tendency to *bleeding* in and around the pancreas and sometimes elsewhere. *Glycosuria* is, on the whole, rare, but with chronic pancreatic disease an excess of sugar in the food may be followed by temporary glycosuria.

Physical Signs.—An enlarged pancreas may often be felt between the epigastric and the umbilical regions, and it may transmit the aortic pulsation. When the stomach is empty there may be dulness on percussion, and palpation becomes easier; the stomach when distended with gas lies in front of and above pancreatic tumors, and gives tympanitic resonance. The colon lies below the pancreatic tumor, and when distended this relation can be easily made out. In acute cases there is very marked tenderness with rigidity of the recti, and some bulging of the upper part of the abdomen. In chronic cases there may be no tenderness and palpation is easier. Pancreatic tumors hardly move with respiration.

PANCREATIC HEMORRHAGE.

This usually is seen in adult males without obvious cause. Arterial or pancreatic disease and trauma are said to predispose to it. There is bleeding into and around the pancreas. Fat necrosis is usually associated.

Symptoms.—In a severe case there is a pancreatic crisis, with subnormal temperature, and some distention of the upper abdomen. The general symptoms of internal hemorrhage may appear, and death may ensue within a few minutes or a day.

The **treatment** is an operation when possible.

Pigmented scars of old bleedings of small size have been found at autopsies, but nothing is known about them in a clinical way.

ACUTE PANCREATITIS.

Etiology.—The usual cause is obstruction of the duct by gall-stones impacted in the diverticulum of Vater and inflammation or tumors at the opening of the duct, which cause regurgitation of bile into the pancreas. Sometimes it follows trauma or bleeding into the pancreas. It is a bacterial infection.

Lesions.—(1) *Acute suppurative pancreatitis:* There is foul pus in the ducts, there may be multiple miliary abscesses throughout the pancreas, or a single large abscess. (2) *Hemorrhagic pancreatitis:* The pancreas is large, shows acute exudative inflammation and hemorrhages which may be large or small, and may involve the tissues around the pancreas. (3) *Gangrenous* or *necrotic pancreatitis* often follows the hemorrhagic form; there are areas of gangrene, large or small, single or multiple; the pancreas is usually enlarged. There are fat necrosis and a local peritonitis in the lesser sac, or a general peritonitis. Gall-stones are often present.

Symptoms.—The *hemorrhagic* and *gangrenous forms* are most acute. The onset is abrupt with a pancreatic crisis; there is usually some fever, which may reach 102°–103° F. After the first day there may be swelling in the epigastrium, and the physical signs of a pancreatic tumor, or merely rigidity and distension. More or less jaundice is common. In the hemorrhagic form the patient dies in from two to five days in collapse or of peritonitis. The gangrenous cases may live one or two weeks and die in collapse.

Abscess of the Pancreas.—The infection is less intense; the symptoms may begin subacutely. After a few days there may be septic fever, diarrhea, and jaundice. The tumor is usually palpable, at least with anesthesia. The abscess may rupture into the stomach, the colon, the peritoneum, or elsewhere.

The **treatment** is surgical and is fairly successful.

CHRONIC PANCREATITIS.

Etiology.—The usual cause is obstruction of the duct, usually by a gall-stone, sometimes from pancreatic stone or a tumor.

Lesions.—(1) *Interlobular type:* New connective tissue forms around the lobules, there is atrophy of the alveolar cells, but not of the islands of Langerhans. (2) *Interacinar type:* The connective-tissue formation is more diffuse with atrophy of the islands also. In both the gland is large, nodular, hard, and all or a part of the gland may be involved. Fat necrosis is rather rare. There are the lesions of an obstructed common bile-duct in most cases.

Symptoms.—There may be attacks resembling pancreatic crises: the stools show absence of pancreatic secretion (p. 200). Rarely glycosuria is present. There is loss of flesh and strength, jaundice is common.

The **prognosis** is not definitely known.

Treatment.—A diet containing less fats. The administration of raw pancreas or pancreatin. An operation when the ducts are obstructed.

PANCREATIC CYSTS.

Causes.—The duct may be obstructed by stone, tumors, or inflammation in or around the duct; or by trauma. A hematoma may form a cyst. Cysts are more common in adults.

Lesions.—The cyst may be small or large. Sometimes it is enormous, holding many quarts. The contents are dark brown, mucoid, alkaline, specific gravity 1010 to 1020, and contain albumin, often blood or pigment, leukocytes, degenerated epithelium, and cholesterin. Ferments often are present. The pancreas usually is atrophied.

Symptoms.—There may be one or several acute attacks like mild crises; the stools may show lack of pancreatic secretion. The tumor grows and when large may cause jaundice, portal obstruction, and a sense of weight or fulness in the abdomen. There is a moderate loss of flesh and strength. *Physical signs:* On inspection bulging may be extreme in the upper part of abdomen. The tumor is tense and elastic, flat on percussion. It has the usual characteristics of a pancreatic tumor (p. 200). Occasionally cysts of the tail of the pancreas lie in the left side of the abdomen, displacing the spleen.

Course.—Usually chronic, in some cases ten or even twenty years. The cyst occasionally gets smaller temporarily or permanently.

The **treatment** is surgical.

CARCINOMA OF THE PANCREAS.

Etiology.—Usually in males from forty to sixty. *Primary* carcinoma is usually adenocarcinoma or scirrhous. The pancreas is large and hard; the growth, usually in the head, often closes the common bile-duct and the pancreatic duct. *Secondary* growths are common.

Symptoms.—Attacks of pain, mild or severe like a pancreatic crisis; stools show lack of pancreatic secretion. Jaundice is intense and persistent; the gall-bladder is usually dilated. Portal obstruction is rare. There may be signs of a pancreatic tumor. The general symptoms of cancer are present. The patient dies in from six months to a year.

Treatment is surgical and symptomatic.

PANCREATIC CALCULI.

These are rare. The stone is usually white and hard, composed of calcium carbonate. In size they vary from fine granules to an inch or more in length. They lodge in the duct and cause interstitial pancreatitis, dilatation of the duct, sometimes a fistula opening into the stomach, the colon, or peritoneal cavity; they may result in pancreatic abscess, and they predispose to cancer.

Symptoms.—Attacks of pain like pancreatic crises. There is no jaundice; pancreatic secretion is absent from the stools; occasionally there is glycosuria; there is moderate loss of flesh and strength.

The **treatment** is operative.

QUESTIONS.

Discuss fat necrosis.

Give the symptoms of a pancreatic crisis and its differential diagnosis from a gastric crisis.

Give the symptoms associated with the absence of pancreatic secretion from the intestines.

Give the physical signs of a large pancreas or pancreatic tumor.

Give the causes, the lesions, and the symptoms of pancreatic hemorrhage.

Give the causes, the lesions, and symptoms of acute pancreatitis?

What are the varieties of acute pancreatitis?

Give the etiology, the lesions, and the symptoms of the two types of chronic pancreatitis.

Give the etiology, the lesions, and the symptoms of pancreatic cysts.

Give the etiology and the symptoms of cancer of the pancreas.

Give the treatment of the preceding conditions.

DISEASES OF THE PERITONEUM.

ACUTE GENERAL PERITONITIS.

Etiology.—*Bacteria:* Usually the pyogenic staphylococci or streptococci, pneumococcus, colon bacillus, typhoid bacillus, or gonococcus often mixed with other forms. (1) *Primary cases* occur in nephritis, arteriosclerosis and gout, but the mode of infection is not known. (2) *Secondary cases* follow: (*a*) a wound (operation); (*b*) perforation of the stomach, intestines, appendix, gall-bladder, or urinary bladder; (*c*) rupture of an abscess of the liver, spleen, kidneys, ovaries, tube, or a lymph-node, etc.; (*d*) from extension of inflammation from any organ covered by the peritoneum or through the diaphragm. The most common cause is perforation in typhoid, gastric ulcer, appendicitis, or dysentery.

Lesions.—The peritoneum is dull and congested. Soon there is an exudate that may be fibrinous, or serofibrinous, or purulent. The pus may be thin and yellowish green, or thick and white or putrid. The effusion following wounds, cancer, or tuberculosis is often bloody. The peritoneal cavity may contain free gas and material from the stomach or intestines. The amount of liquid varies from a few ounces to several quarts. The intestines are distended and often matted together.

Symptoms.—With *perforation*, if the patient is not too weak, there is a sudden, sharp, severe pain at about the site of rupture. This may be colicky or constant, it may be transient or protracted. If fever is present the temperature may drop 2 to 4 degrees. There is more or less collapse. *Other cases* may begin with chills and a rapid rise of temperature. *In about a day* the symptoms become marked. The tongue *is coated, and later* dry and glazed. There are hiccough, eructations, vomiting, often repeated. The vomitus becomes yellowish, greenish, or dark, and may have a fecal odor. There are constipation and tympanites; abdominal pain of varying intensity; a rapid, feeble heart, a rapidly increasing leukocytosis; rapid, shallow, painful, costal breathing; a remittent or continuous temperature, 103°–105° F. Prostration is very marked. Toward the end the vomiting stops; delirium, stupor, and then coma develop, and the patient dies. *Physical examination:* The face is dusky, the expression pinched and anxious, the skin is cold and clammy. The patient usually lies with the thighs flexed; the breathing is thoracic; the heart apex may be in the fourth space, the pulse very small, rapid, and wiry, but later soft. The abdomen may be hard and retracted, but usually there is increasing distension and marked tympanites. It is very tender. The liver dulness in the nipple line and the splenic dulness are often absent with perforation. The signs of ascites are usually present. *Asthenic cases* in those already very ill may be too insidious for diagnosis; the patient grows sicker and dies in collapse.

Sometimes *primary cases* recover, but the other cases die in one to seven days unless operated upon early.

LOCAL PERITONITIS.

(1) **Subphrenic abscess** is a collection of pus between the liver and the diaphragm, pushing the diaphragm upward. It is due to extension of inflammation from the pleura, liver, or gall-bladder. The symptoms resemble empyema.

(2) **Subphrenic pyo-pneumothorax** is a collection of pus and gas between the liver and diaphragm, more often on the left side. It usually follows perforation of the stomach. The onset resembles acute general peritonitis except that the pain and tenderness are in the upper part of the abdomen and the thorax. There are the physical signs of pneumothorax over the lower part of the chest and of compressed lung over the upper part of the chest. After a few days the general symptoms are those of septicemia. The patient may live from one to several weeks.

(3) **Peritonitis of the lesser sac** may follow perforation of

the stomach, colon, duodenum or extend from the pancreas. The subjective symptoms resemble general peritonitis. The local symptoms are those of a tumor in the epigastric region, which is tender, dull on percussion, and elastic. Below it is the colon, at its left the spleen. The stomach when distended with gas obliterates the upper part or all of the dulness. The exudate usually ruptures into the general peritoneal cavity with fatal result.

(4) The most common forms of local peritonitis are due to inflammation of the appendix or female pelvic organs.

(5) There may be a fibrinous peritonitis over any viscus with local pain, tenderness, and crepitations.

Treatment of Acute Peritonitis.—Acute general peritonitis and all cases of localized peritoneal abscess are surgical, but perhaps cases due to the gonococcus, pneumococcus, and colon bacillus or the primary cases may recover without laparotomy. The *medical treatment* consists of absolute rest in bed; liquid diet by mouth or rectum; morphin hypodermically for the pain and ice on the abdomen continuously. *For vomiting:* Lavage, rectal feeding, cracked ice in the mouth, sips of champagne, and the usual anti-emetics. *For tympanites:* The rectal tube, turpentine stupes, turpentine enemas. *For constipation:* High large enemas. *The saline treatment:* Epsom salts every morning to give three or four loose stools daily. This should lessen the chance of adhesions, tympanites, and constipation, and should promote recovery.

QUESTIONS.

Give the etiology, lesions, symptoms, and treatment of acute general peritonitis.

Give the causes, lesions, and symptoms of subphrenic abscess.

Give the causes, lesions, and symptoms of subphrenic pyopneumothorax.

Give the causes, lesions, and symptoms of peritonitis of the lesser sac.

CHRONIC PERITONITIS.

There may be **local adhesions** around any abdominal viscus. Often this causes no symptoms, but there may be local pain or persistent intestinal colic. The bands may cause intestinal obstruction.

Diffuse adhesive peritonitis follows acute peritonitis, simple or tuberculous, and results in the general replacing of peritoneum by connective tissue, which mats all the viscera together.

Chronic proliferative peritonitis may be found in alcoholism, cirrhosis of the liver, ascites, and chronic portal congestion. Masses of thick connective tissue form on the peritoneal surfaces of the organs, and may lead to contraction of the liver, spleen, omentum, etc. Adhesions usually are few and not marked. Ascites is often present. A friction rub often is felt and heard.

Treatment.—Build up the general health by fresh air. Treat the ascites. Counterirritation may be used over local peritonitis. A snug elastic abdominal binder worn for months may help the more extensive forms.

QUESTION.

Discuss chronic peritonitis.

TUMORS OF THE PERITONEUM.

Etiology.—Primary tumors are rare. Usually they are endothelioma or carcinoma (especially colloid of the omentum). Secondary tumors are common. They are usually secondary to tumors of abdominal or pelvic viscera. Miliary or large masses are seen throughout the peritoneum. The colloid carcinoma of the omentum forms a very large elastic mass, weighing several pounds.

Symptoms.—There are the general symptoms of cancer, the special symptoms of the primary tumor; and locally the symptoms and signs of ascites and the signs of the tumor masses. Ascites usually is absent in colloid of omentum.

QUESTIONS.

Give the causes and symptoms of new growths of the peritoneum.

What is the usual form of primary peritoneal tumor and its symptoms?

ASCITES.

Etiology.—(*a*) *Local causes* are: (1) Portal obstruction from any cause; (2) inflammations or tumors of the peritoneum, or other abdominal tumors; (3) obstruction of the lymphatics; (4) perforations. (*b*) *General causes:* Cardiac, renal or blood conditions, causing dropsy elsewhere also.

Lesions.—There is liquid in the peritoneal cavity. It is usually clear and yellow, but may be stained with bile or blood or may be chylous and milky. It may have the characteristics of an exudate or a transudate. The amount varies, it may be several gallons. The peritoneum usually looks normal, but may be thickened or inflamed.

Symptoms.—The onset usually is slow. The *subjective symptoms* are indigestion, constipation, increasing weight and fulness in the abdomen, pain in the loins, dyspnea on exertion or lying down, perhaps syncope. There is a gradual loss of strength. The urine is usually lessened, and may be albuminous. *Physical signs:* The apex-beat may be in the fourth space; the abdomen is large and prominent, especially in the dependent parts. The navel may protrude; the lower ribs are everted. Striæ or the caput medusæ may be prominent. There may be edema of the legs. The fluid percussion wave is present. *Percussion:* When the patient is lying down there is an elliptical area of resonance around the navel, with flatness in the flanks that is continuous with the dulness of the liver and spleen. These signs change with change of position, the resonant area always being uppermost. The upper limit of the hepatic or splenic dulness may be raised a space or two. An aspirating needle inserted over the area of flatness withdraws the liquid.

The **prognosis** and **course** vary with the cause. If this be removable the liquid may be absorbed.

Treatment.—*Paracentesis:* The bladder must be empty. After cleaning the skin a trocar and canula is inserted in the linea alba in the hypogastric region, and the liquid is allowed to run out. A tight abdominal binder is then applied. After paracentesis, salines may be given to cause three or four loose stools a day, and liquids must be restricted. If the liquid reaccumulates, tapping may be repeated. The primary condition may need treatment

QUESTIONS.

Give the etiology, lesions, symptoms, and treatment of ascites.

How would you differentiate ascites due to inflammation from dropsy of the peritoneal cavity?

DISEASES OF THE RESPIRATORY ORGANS.

HAY FEVER.

Etiology.—Three factors coexist: (1) A neurotic susceptibility which is often hereditary. (2) Some nasal abnormality, with areas of hyperesthesia. (3) An exciting cause which may be pollen of certain plants (rag weed, golden rod, etc.); odors (rose); animal emanation (from a horse, dog, etc.), or many other things. In a given individual one cause is usually specific. Hay fever may come at a certain time of the year, or may be induced by emotion, exhaustion, etc.

The **symptoms** begin abruptly, the eyes are congested and watery; there is sneezing and a thin nasal discharge. Some cases have with or instead of this, bronchial asthma, pharyngitis, or conjunctivitis. Usually there is much mental and physical depression. An attack lasts from a few hours to a few weeks.

Treatment.—*Preventive:* This includes tonics for the neurotic temperament and the treatment of the nose with the destruction of the hypersensitive areas. The exciting cause should be avoided by a trip at sea, or to the mountains at the proper season, or in other ways. In an attack local application to the nose of suprarenal gland or cocaine is used, and also the treatment for asthma (p. 216).

QUESTION.

Give the etiology, symptoms, and treatment of hay fever.

DISEASES OF THE LARYNX.

Acute catarrhal laryngitis follows exposure to cold or wet; irritating vapors or dust; corrosive or hot liquids; overuse of the voice; it may occur in acute infections or with inflammations of neighboring mucous membranes. A predisposition exists in some persons. It is more common in adults. The *lesion* is an acute catarrh of the larynx (p. 18). The *symptoms* are tickling in the larynx, a dry, croupy cough, but later some mucopurulent sputum; hoarseness, or even aphonia; pain in the larynx on speaking or coughing. In adults with edema, or in babies there is dyspnea. The vocal cords and the rest of the larynx are very red and somewhat swollen. There may be more or less fever for a few days. The *local treatment* consists of steam inhalation, alkaline or astringent sprays, and ice on the neck.

Chronic catarrhal laryngitis may follow acute attacks; long continued irritation; bad air, mouth breathing, or chronic alcoholism. The *symptoms* are like the acute cases, but are less intense and occur in exacerbations, followed by remissions. There is less redness and swelling. *Treatment:* The cause should be corrected, general tonics, local sprays and cold affusions to the neck and chest are indicated.

Edema of the larynx may occur with (1) *inflammations*—acute laryngitis, quinsy, diphtheria, measles, scarlatina, erysipelas, or cellulitis of the neck, chronic ulcers of the larynx, and (2) *without inflammation* in nephritis; with edema elsewhere; or angioneurotic edema. There is edema over the epiglottis, the aryepiglottic folds, or elsewhere in the larynx, with narrowing or obliteration of the lumen. The *symptoms* often begin abruptly and within a few minutes or hours there are hoarseness, aphonia, and often asphyxia and death. Sometimes the edema disappears after a few hours or a day or two. *Treatment:* Ice externally and in the mouth. The swollen tissue may be scarified. Tracheotomy may be needed. The causative condition should be treated.

Laryngismus Stridulus (**Spasmodic Laryngitis**).—This is not an inflammation, but a neurotic spasm of the adductors of the vocal cord. It occurs in children from six months to five years old with rickets. It may be induced by passion.

Symptoms.—There are two groups of cases: (1) The pure neurosis. Upon waking the child sits up, stops breathing, struggles for breath, grows cyanotic, and may have a general convulsion. After several seconds the spasm relaxes and the attack is over. It may be repeated many times within a day or two. It is very seldom fatal. (2) A slight laryngitis is present. The child awakens at night in an attack. He has a husky voice, croupy cough, dyspnea, stridulous breathing, and cyanosis. The attack stops suddenly after an hour or two and the child sleeps. The attack is repeated at about the same hour for three or four nights. In the interval the child is quite well or has a mild laryngitis. It is never fatal.

Treatment.—(1) A warm bath (90°–105° F.) with cold affusions to the head and chest, or pushing a finger into the child's throat may stop the spasm. (2) Cause vomiting by titillating the epiglottis or by emetics. The bath, chloroform inhalation, a croup-kettle, or ice or mustard to the neck, may be used.

Tubercular Laryngitis.—*Etiology:* It is rarely primary. Usually it is secondary to lung tuberculosis. *Lesions* (see p. 106): The tubercle tissue appears in the subepithelial tissue over and between the arytenoids, aryepiglottic folds, and epiglottis. Later it involves the epithelium and forms ulcers. The cartilages may necrose. *Symptoms:* The voice tires easily, then hoarseness and aphonia develop. There may be pain on swallowing and painful cough. *Laryngoscopic picture:* In the prodromal stage there is anemia of the mucous membrane; later swelling at the interarytenoid space, or elsewhere in the larynx; still later worm-eaten ulcers appear. There may be paralysis of one or both vocal cords. There is a mucopurulent secretion. The swelling may cause dyspnea. *Prognosis* is bad, but cures are reported. *Treatment:* General treatment for tuberculosis. *Locally:* Steam inhalations with benzoin, creosote, or pine oil; insufflation of iodoform; lactic acid as a caustic to ulcers; with dysphagia, cocain before eating. For dyspnea tracheotomy may be needed.

Syphilis of Larynx.—*Etiology.*—It is a secondary or tertiary lesion. In the secondary stage there may be hyperemia with ulcers or condylomata. In the tertiary, hyperemia followed by progressive ulceration which may involve cartilages. Scarring may be followed by stenosis and other deformity. *Symptoms (secondary type):* Alteration or loss of voice and pain in swallowing are present. These pass away in a few weeks. *Laryngoscopic picture:* Congestion and ulceration are more often on the vocal cords; mucous patches or condylomata usually on the ventricular bands or between the arytenoids or on the epiglottis. Secretion is scanty. The *tertiary type* usually starts on the epiglottis. The ulcers are deep with overhanging thickened edges and are yellowish and sloughy. They are surrounded by a zone of inflammation. After healing various deformities appear. *Prognosis:* Secondary cases recover. Tertiary usually recover, but may have edema of the larynx or the effects of the deformities. *Treatment:* Ulcers are treated with silver nitrate or iodoform; stenosis by intubation or tracheotomy.

QUESTIONS.

Give the etiology, lesions, symptoms, laryngoscopic picture, and treatment of acute and catarrhal laryngitis.

Give the causes, symptoms, and treatment of edema of the glottis.

Give the causes, symptoms, and treatment of laryngismus stridulus.

Give the appearances, the symptoms, and prognosis of tubercular and of syphilitic laryngitis.

DISEASES OF THE BRONCHI.

ACUTE BRONCHITIS.

Etiology.—A predisposition exists in some persons; it is sometimes epidemic; is more common in bad weather, with an indoor life; it may follow exposure to cold and wet, or the inhalation of irritating vapors or dust; it is a part of measles, pertussis, influenza, typhoid, tuberculosis, etc. It often follows acute inflammation of the throat and nose. The *lesion* is an acute catarrh of the trachea and bronchi.

Symptoms.—A few days after the onset of the coryza or sore throat the bronchitis develops. There are a sense of oppression in the chest, a dry cough, causing pain in the sternal region and along the attachment of the diaphragm; after two or three days the cough is softer and not painful. The sputum at first is very scanty, mucous, and viscid; later, more abundant, mucopurulent or purulent, yellow or green, and contains pus, degenerated epithelium, dust grains, and bacteria. *Physical signs:* Bronchial fremitus may be felt with breathing. Occasionally resonance at the bases is impaired. On auscultation there may be harsh breathing, sibilant and sonorous and later mucous râles. The general symptoms vary much. At times there is fever, even up to 103° F.; it falls by lysis within a week. The cough persists for a week or two longer than the general symptoms. In *young children* there may be an accumulation of secretion in the bronchi, with dyspnea and cyanosis or bronchopneumonia. In the *weak* and *aged* there may be bronchopneumonia or a protracted course.

Treatment.—An attack may sometimes be aborted by a hot bath followed by a warm bed, Dover's powder gr. x. and quinine gr. x. In the early stage counterirritants to the chest, steam inhalation, potassium citrate, liquor ammonii acetatis, tartar emetic, spiritus etheris nitrosi, or codein may relieve. Later, drugs are less useful, but senega, squills, ammonium carbonate or chlorid, nux vomica, and many others are used. Some cases need stimulation. A child with cyanosis should be made to vomit. With fever the patient should stay in bed.

QUESTION.

Give the etiology, lesions, symptoms, and treatment of acute bronchitis.

CHRONIC BRONCHITIS.

Etiology.—It is found after repeated acute attacks, with chronic lung diseases, with inhalation of dust, chronic heart disease, aortic aneurysm, gout, nephritis, and in the aged.

Lesions.—It is a chronic catarrh (see p. 18). The mucous membrane may be thick and hypertrophied, or thin and atrophic. There may be bronchiectasis. Usually emphysema is present.

Symptoms.—(1) **The Ordinary Form.**—General symptoms are slight or absent. There is cough, especially in the morning or during the night; the sputum is fairly abundant, mucopurulent, and yellowish or green. The *physical signs* are as in acute bronchitis. The symptoms and signs of the associated conditions are very important.

(2) **Bronchorrhea.**—The sputum is very abundant, thin

and serous, or thick and purulent. Bronchiectasis and fetid bronchitis are common sequels.

(3) **Fetid or Putrid Bronchitis.**—The sputum is abundant and fetid. Three layers form upon standing—an upper layer of dirty froth, a middle layer of opalescent liquid, and the lowest layer, a granular sediment, contains Dittrich's plugs (yellow masses containing mucus, bacteria, pus, detritus, granules and crystals of fat). The general health suffers, and there are anemia, loss of flesh and strength, and fever. The febrile exacerbations may last two or three weeks and be well marked. In the following remission the sputum is less abundant, but has the same characters. Pneumonia, abscess or gangrene of the lung, ulceration of the bronchi, bronchiectasis or cerebral abscess may occur.

(4) **Dry Bronchitis.**—There are severe attacks of coughing, with scanty and viscid sputum. It is found in the aged with marked emphysema. The signs are those of emphysema, with sibilant and sonorous râles. All cases have summer remissions and are worse in winter.

Prognosis.—Cure is rare in older persons, but much relief may be obtained.

Treatment.—Avoid bad weather and live out of doors in an equable warm climate. Tonics and treatment of the associated conditions are needed. In an exacerbation potassium iodid, gr. xxx a day for many weeks, or combinations of codein, ammonium chlorid or carbonate, senega, strychnin, turpentine, balsams, and creasote may help. For the dry form steam inhalation is used. For the fetid form, sprays and steam inhalation with carbolic acid, turpentine, or eucalyptol in the water.

QUESTIONS.

Give the etiology and lesions of chronic bronchitis.

Give the varieties, the symptoms of each, and the differential diagnosis.

What is the prognosis and treatment?

BRONCHIECTASIS.

Etiology.—This may follow any chronic lung disease, foreign bodies in the bronchi, pertussis, pressure on a bronchus from aneurysm or tumor, or thickened pleura. Rarely it is congenital. The bronchial wall is weakened and then stretched by the weight of accumulated secretions, by pressure of the air in coughing, or it is pulled out by contraction of connective tissue. Usually it occurs in adult males.

Lesions.—Dilatations of the bronchi are present and may be cylindrical or sacculated. *Universal bronchiectasis* involves one lung. The bronchi become a chain of sacculi of any size up to three or four inches. The *partial form* is more common. There are sacculated or cylindrical dilatations in one or both lungs. The dilatation contains a variable amount of secretion which may be fetid. There may be ulceration of the dependent parts. *Microscopically:* The wall of the bronchus is thinned, the epithelium flat, the muscle and elastic layers stretched and often incomplete. There are the associated lesions.

The **symptoms** are often masked by the primary condition, but a large sac may be recognized. There are severe attacks of coughing at intervals of several hours, especially in the morning and upon change of position. Sputum amounts to several ounces, and resembles that of fetid bronchitis, but the fetor is often absent. Sometimes there is hemoptysis or cyanosis on exertion. The physical signs of a cavity (see p. 114) without a surrounding zone of consolidation are present usually at the base. Impaired health and septic symptoms are present. It lasts years and is incurable.

Treatment.—Sedatives and ordinary expectorants are useful. A superficial cavity may be incised and drained.

QUESTION.

Give the causes, lesions, and symptoms of bronchiectasis.

BRONCHIAL ASTHMA.

Etiology.—A predisposition is hereditary. Asthma is more common in neurotic persons, especially males, and may follow neuralgia, epilepsy or pertussis. The exciting cause may be the air of some special place, dust, odors, pollen, animal emanations, excessive emotion, a certain article of diet, overeating or indigestion, perhaps disturbances of the tubes or ovaries. One of these is usually specific in a given person. Many cases are a part of hay fever (see p. 209). The attack is supposed to be due to (1) spasm of the muscles of the bronchi; or (2) swelling from hyperemia of the mucosa; or (3) a special form of exudative inflammation of the bronchioles. There is a peculiar, scanty, mucous secretion. Later emphysema or a large right heart are apt to be present.

Symptoms.—Prodroma are often present for a few hours, such as chilly feelings, tightness in the chest, flatulence, polyuria, headache, vertigo, and marked depression of spirits. The attack, often at night, comes on rapidly. There are pallor, sweating, some cyanosis, a sense of suffocation, intense dyspnea, rapid, weak, small pulse, a normal or subnormal temperature, and marked eosinophilia. *Physical signs.—Inspection:* The shoulders are raised and fixed; accessory respiratory muscles act forcibly; the chest moves but little, and seems fixed in full inspiration; inspiration is short; expiration much prolonged but ineffective. *Percussion* gives a normal or hyperresonant note. *Auscultation:* Inspiration is short and feeble, expiration very long; sibilant and sonorous râles, and later moist râles are heard. Cough is violent at first. The sputum consists of gelatinous translucent masses that may be shaken out into casts of small bronchi, and contains Curschmann's spirals. These are small cylinders with a very refractive thread of mucin wound into a helix, they also contain eosinophiles and Charcot-Leyden crystals. After a day or two the sputum is mucopurulent.

Course.—The attack lasts a few minutes or hours, usually stops suddenly, and is apt to recur on the next two or three nights. In the remissions there may be some wheezing and slight cough or the patient may be quite well. After a few years chronic bronchitis or emphysema is apt to develop.

Prognosis.—The attacks are not fatal. They may cease altogether after a few years or in a proper climate.

Treatment.—Avoid the exciting cause. Everyone may find some place where he will be free. Potassium iodid, gr. xxx a day for months, may cure. General hygienic treatment is important.

In the attack remove the offending flowers, etc. *Inhalation of chloroform,* ether, amyl nitrite, oxygen, compressed air, the fumes of niter-paper, stramonium or hyoscyamus leaves, asthma cigarettes or tobacco; or taking by mouth coffee, alcohol, chloroform, suprarenal, morphin and atropin may relieve. The treatment of hay fever may be needed.

QUESTION.

Give the etiology, the symptoms, including the characteristics of the sputum, and the treatment of bronchial asthma.

FIBRINOUS BRONCHITIS.

This is a rather rare condition, more common in males; in the spring; with tuberculosis or chronic pleurisy; or skin diseases. There are attacks of coughing with the expectoration of a white, opaque mass that, when shaken out, is seen to be a complete cast of part of the bronchial tree. It is solid or tubular, is composed of fibrin or mucin, leukocytes, and Charcot-Leyden crystals. The tips of the branches may resemble Curschmann's spirals. These attacks recur at various intervals and are accompanied by more or less constitutional disturbance.

QUESTION.

Describe fibrinous bronchitis.

DISEASES OF THE LUNGS.

CIRCULATORY DISTURBANCES.

Active Congestion.—An increased blood flow and dilatation of the pulmonary vessels occurs around any acutely inflamed part of the lung, with inhalation of hot, very cold, or irritating air, violent exertion, or acute alcoholism. Marked cases may have dyspnea, cough, and bloody frothy sputum. *Physical signs:* At both bases behind there are increased fremitus, some dulness, feeble or bronchovesicular or even bronchial voice and breathing, and perhaps moist râles. After a few hours it ends in recovery or edema or pneumonia. *Treatment:* Cup and counterirritate the chest behind, use venesection (10–30 oz.), and perhaps veratrum or nitroglycerin.

Passive Congestion.—(1) **Mechanical Congestion** (brown induration, pneumonia of heart disease).—This is caused by protracted obstruction or increased tension in the vessels of the lungs in mitral lesions, right heart weakness, or pressure on the veins. *Lesions:* The lungs are large, reddish brown, heavy, less crepitant, are tough on cutting, and the surface changes color in the air to bright red. The changes begin at the bases and extend to the whole of the lungs. *Microscopically* there are dilated capillaries; chronic productive inflammation of the stroma of the lung; the alveolar cells are swollen, pigmented, and desquamated. *Symptoms* are absent until compensation fails, then there are dyspnea, cough, and sputum that contains pigmented epithelium, and may be bloody or frothy. Treat the cause.

(2) **Hypostatic Congestion.**—(*a*) May occur in any bedridden person who has a weak heart; (*b*) to a slight extent it may be due to anything that displaces the diaphragm upward; (*c*) it is found in brain diseases or injuries. *Lesions:* The dependent part of the lungs (posterior borders at the base) are dark red, firmer, and when cut drip bloody liquid. There is an exudate in the air spaces and stroma in the severe cases (splenization or hypostatic pneumonia). The *symptoms* are very obscure, the patient gets weaker and often dies. *Physical signs* are the same as in active congestion. *Treatment:* With heart disease bleed (10–30 oz.) and use cups and counterirritation. It may often be avoided by frequent change of position.

Edema of the Lungs.—(*A*) **Collateral edema** is a local edema around the site of acute inflammation in a lung. The additional symptoms due to it are cough and frothy sputum, with signs of incomplete consolidation and moist râles.

(*B*) **General Edema.**—*Etiology:* Similar to congestions. It is a common antemortem change in cachexias; anemia; heart, lung, kidney, or acute infectious diseases; and in comas. *Lesions:* The edematous part of the lung is heavy, but floats; it pits on pressure; the cut surface may be translucent, and froth, sometimes bloody, may be squeezed from it. There is transudation into the stroma and air spaces. Congestion is often marked. It begins at the bases behind, but may involve all of the lungs. *Symptoms:* To the symptoms of the primary disease are added cough, with frothy abundant sputum, sometimes bloody or even tenacious; a progressively faster and weaker heart, cyanosis, cold livid limbs, and usually death. *Physical signs* are like congestion, but the moist râles are very distinct. The *prognosis* depends largely on the primary condition, but is rather bad. *Treatment* is practically that of hypostatic congestion.

Bronchopulmonary Hemorrhage (Hemoptysis).—*Etiology:* (1) Healthy adolescents may have one attack without sequels. (2) It may follow violent muscular exertion, or blows on the thorax, or lung diseases, such as tuberculosis, intense congestion, pneumonia at the onset, cancer, gangrene, abscess, or bronchiectasis. (3) It may come from ulcers in the larynx, trachea, or bronchi, and may be profuse. (4) Heart disease, especially mitral. (5) Aneurysms (small repeated bleeding for weeks, or sudden rupture). (6) Vicarious menstruation; arthritic hemoptysis; pulmonary endarteritis; malignant fevers and hemorrhagic conditions. (7) Parasitic hemoptysis (see p. 124). (8) The blood may come from the mouth or pharynx. *Lesions:* Bleeding may be from capillaries or from larger vessels. The lung may be pale or may contain little clots in the air spaces. There are the associated lesions. *Symptoms.—Mildest cases:* A little blood is seen in the sputum. *Moderate cases:* The patient has a warm, salt taste in the mouth for a few seconds, then coughs and spits up blood. It is bright red, frothy, alkaline, seldom clotted, and is mixed with sputum. It may amount to an ounce or two or to a pint; the bleeding may be repeated for a few days. After bleeding stops the sputum is blood-stained for three or four days. *Severer cases:* With coughing repeated mouthfuls of blood are spit out, and soon there are symptoms of the loss of blood or the patient may drown from filling of the bronchi and lungs. Some blood may be swallowed and vomited later, or after a day or two the stools may be tarry. Usually the patient is much frightened and restless. The bleeding, unless from an eroded artery or aneurysm, stops spontaneously. *Physical examination* is dangerous, but moist râles may be heard. Some cases are quickly fatal. *Treatment: Mild* or *moderate cases* need rest for mind, heart, and body. The patient is put in bed and kept there for three days after the bleeding stops. Morphin is given hypodermically. For an overacting heart, an ice-bag to the precordium or affected part of lung. Very little food should be given. With *serious loss of blood* ligate three limbs at once at the hip and shoulder, so as to constrict the veins, but not the arteries ; every fifteen minutes release one limb and tie the free limb in regular order. Give no morphin or stimulant to these cases.

Pulmonary Apoplexy.—(1) **Diffuse.**—There is extravasation of blood into a lung, with laceration of the lung. It occurs with ruptured aneurysms, incised or lacerated wounds of the lung, and sometimes in sepsis. *Symptoms:* Profuse hemoptysis, dyspnea, collapse, and physical signs of consolidation. It is apt to be fatal from loss of blood, or subsequent infection.

(2) **Pulmonary embolism** or **thrombosis** is usually seen in heart disease, but may occur in infectious diseases. *Lesions:* There are one or more wedge-shaped areas, firm and red, with the artery at the apex of the wedge plugged with an embolus or thrombus. The pleura involved is inflamed. The air spaces and stroma of the infarct contain blood. Later this changes to a puckered brown scar, or it may slough and lead to abscess, gangrene, or pneumothorax. *Symptoms:* Often there are a sudden pain in the side, cough, hemoptysis, dyspnea, perhaps syncope or convulsions, and fever. *Physical signs* of consolidation, with pleural râles, are present, usually over the base behind. Large infarcts may cause urgent dyspnea for a few minutes, collapse and death. *Treatment:* Rest and symptomatic treatment.

QUESTIONS.

Give the causes and symptoms of active congestion of the lungs.

Give the causes and symptoms of passive congestion of the lungs.

Give the causes, symptoms, prognosis, and treatment of edema of the lungs.

Give the causes of hemoptysis.

Differentiate hemoptysis from hematemesis.

Give the treatment of hemoptysis.

BRONCHOPNEUMONIA.

Etiology.—*Bacteria:* Pneumococcus, Streptococcus pyogenes, and Staphylococcus pyogenes aureus are most important. Primary cases are usually due to the pneumococcus. These bacteria occur alone, combined with one another, or with other bacteria. *Primary cases* are caused like lobar pneumonia, but are usually found in children under five. *Secondary* bronchopneumonia is more common. It occurs (1) with infectious diseases, ileocolitis, rickets, and marasmus. (2) As aspiration pneumonia in coma, after anesthesia, operations on the mouth or upper air-passages, tracheotomy, cancer of the larynx or esophagus, hemoptysis, bronchiectasis, tuberculosis of lung, etc. (3) In weak, bedridden old persons. In general it is more common in children and old persons; in institutions; with previous ill-health and unhygienic surroundings; poor food and air; after exposure; and in winter and spring.

Lesions.—The lungs show slightly depressed, bluish areas of collapse, and slightly elevated areas of consolidation with roughened pleura. They are heavier and resistant. The cut surface shows consolidated areas of varying size and shape that may run together. These are hard, red or gray, and not granular. They surround the bronchi. Around and beyond them may be collapse or splenization. Usually they are at the back of the lungs and in the lower lobes. There is also bronchitis. The bronchial nodes are enlarged. *Microscopically,* the characteristic lesion is a productive inflammation in and around the bronchus, associated with exudative inflammation of contiguous lung. The air spaces and bronchus are plugged with an exudate of mucus, pus, epithelium, and bacteria. Different areas show all stages of the process. There is more or less emphysema of the rest of the lung. The exudative inflammation resolves. The new connective tissue is permanent, although it may not appreciably damage the lung.

Symptoms.—In **children**: *Primary* cases and *secondary* cases in patients not too weak have an abrupt onset or follow bronchitis. A chill is rare. There are fever, prostration, and cough. There may be vomiting, diarrhea, tympanites; rapid pulse (150–200), growing feeble; leukocytosis, cyanosis; respirations 50–80; real dyspnea; cough, often severe, but sputum is not seen except with vomiting; it is mucopurulent and often blood-streaked. The temperature has a daily maximum of 103°–106° F. and is remittent or even intermittent. Cerebral symptoms are often severe, and may even suggest meningitis. Prostration is marked, and the typhoid state often develops. *Physical signs:* Besides those of bronchitis, there are areas over both lungs, especially behind and over the lower lobes, of more or less irregular signs of consolidation, such as increased fremitus; dulness; breathing and voice feeble, harsh, bronchovesicular, or even bronchial; râles moist, and fine or coarse; and sometimes friction sounds. Old areas fade and new ones appear as the disease continues.

Course.—The symptoms persist for one to four weeks and go by lysis. There may be striking remissions for a day or two, followed by exacerbations or relapses. *Acute congestive cases* have a very intense onset and death in one to five days. *Protracted cases* run on for four to eight weeks, with periods of a day or two without fever. In *weak children* there are merely rapid breathing, perhaps fever, and death in a few days.

In **adults** the symptoms are similar, but the signs are apt to be more typical.

The **aged** become weak and die in a few days. There may be no fever or cough. The signs are usually present, but incomplete.

Complications are like those of lobar pneumonia, but abscess, gangrene, and meningitis are more common. Tuberculosis may complicate, but these cases are usually tuberculous from the start. There may be tuberculosis of the bronchial nodes.

The **prognosis** depends on the intensity of the disease and the previous state of the patient. Primary cases in children often recover. In institutions it is much worse. Inhalation pneumonia and cases in the aged are very serious.

Treatment.—*Preventive:* Care of the mouth, frequent change of the position of the bedridden, the avoidance of exposure in convalescence from infectious diseases. The pneu*monia* is treated like severe bronchitis and lobar pneumonia.

QUESTION.

Give the etiology, lesions, symptoms, prognosis, and treatment of bronchopneumonia.

CHRONIC INTERSTITIAL PNEUMONIA.

Etiology.—The **local form** is associated with tuberculosis, emphysema, gummata, hydatids, or a thick pleura.

The **diffuse form** follows lobar pneumonia (rare), bronchopneumonia, chronic pleurisy, pneumonoconiosis, or atelectasis from pressure of an aneurysm or other tumor.

Lesions.—In an advanced diffuse case, one lung is very small, tough, largely airless, and solid. Chronic bronchitis, bronchiectasis, and a thick pleura are usually present. *Microscopically:* Firm fibrous tissue is abundant. The other lung and the nonfibrous parts of the affected lung are emphysematous. The heart is pulled toward the affected side; the right ventricle is much hypertrophied; there is pulmonary arteritis.

Symptoms.—There is a very slow onset of dyspnea on exertion and the symptoms of chronic bronchitis and bronchiectasis. *Physical signs in a marked case:* The affected side of the chest is diminished in girth; does not expand in breathing; is retracted; the ribs are close together; the shoulders droop, and scoliosis is present. Fremitus is increased or, less often, diminished. There is dulness or flatness. Bronchiectasis may give tympany or an amphoric note. Breathing and voice are feeble, or bronchial or cavernous. Râles are present—sibilant, sonorous, moist and fine, or coarse and gurgling; pleural frictions and creaking. The other lung gives signs of emphysema. The heart gives the signs of displacement and of the changes in the right ventricle.

Prognosis.—They live ten or twenty years, perhaps in fair health, but are apt to die of pneumonia of the other lung, of tuberculosis, or of the heart lesion.

Treatment.—An equable climate is helpful. The associated conditions should be treated.

PNEUMOCONIOSIS.

Etiology.—It follows the protracted inhalation of dust that acts mechanically rather than chemically, such as coal (anthracosis); stone (chalcicosis); metallic dust (siderosis), especially in grinders; dust from grains, cotton or linen, cement, etc. It is an occupation disease.

Lesions.—The lungs are large and emphysematous, with chronic bronchitis. In anthracosis they are black. Connective tissue is seen in strands, or diffusely throughout the septa or alveolar walls and in the bronchial walls. The lymph-nodules and nodes are large and sclerosed, and in anthracosis are black. Later the lymphoid tissue tends to necrose and form little cavities or ulcers. *Microscopically*, the dust particles are seen in the bronchial and alveolar epithelium; in the lymph-nodules in the lung; bronchial, tracheal, and substernal nodes; in lymph-vessels leading to them, and in the interlobular connective tissue, especially near the pleura. Occasionally they run this gauntlet and are found in the liver and spleen also.

The **symptoms** and *physical signs* are those of emphysema, chronic bronchitis, and bronchiectasis. The sputum contains the dust grains in the epithelial cells and leukocytes. In anthracosis it is black or dark gray. The patient is very apt to die of lung tuberculosis.

Treatment for the associated conditions is indicated, but *prophylaxis* is all important. This consists in good ventilation of the working places and in special apparatus to carry off the dust and prevent its inhalation.

EMPHYSEMA.

(1) **Interstitial** or **interlobular emphysema** is due to rupture of the lung from a penetrating wound (fractured rib), violent cough or other muscular strain which results in the escape of air, as fine or large beads, into the connective tissue between the lobules. It may extend to the trachea, or neck, or result in pneumothorax. The process may extend from a wound in the neck, especially tracheotomy, to the lungs.

(2) **Vesicular Emphysema,—Compensatory.**—If part of the lungs is not functionally active the rest of the lungs expands more fully. This occurs in pneumonias, tuberculosis, pleural effusions or adhesions, pneumothorax and interstitial

pneumonia. If the condition persists long enough substantive emphysema follows.

(3) **Hypertrophic, Idiopathic or Substantive Emphysema.**—*Etiology.*—It is due to weakness of lung tissue and excessive air pressure in expiration. It is more common during and after middle life, but is not rare in children; more common in males; heredity is important. It follows chronic cough, as in pertussis, adenoids, asthma, chronic bronchitis; is common in horn players, glass-blowers, and very hard muscular workers; and in heart disease, especially mitral.

Lesions.—Rib cartilages are calcified. The lungs quite hide the pericardium, are large, pale, soft, dry, less elastic and pit on pressure; the air sacs are large, and may form protruding bleb-like areas. The change is most marked in front and at the root. The pleura is pale, and may show unpigmented areas (albinism). *Microscopically*, the air-passages and cells are dilated; their walls thinned and may contain holes or be wholly absorbed. There is less elastic tissue, the capillaries atrophy, the epithelium is degenerated and more or less atrophic. There is new connective tissue in the interlobular septa and around the bronchi and blood-vessels. There is chronic bronchitis, and perhaps bronchiectasis. The diaphragm is depressed. The results of chronic congestion are seen in the heart, liver, and kidneys.

Symptoms.—The onset is slow. There are dyspnea on exertion; later constant dyspnea or orthopnea, marked cyanosis, and the symptoms of chronic bronchitis; the cough may become constant. Attacks like asthma are common. The temperature is often subnormal; there is a slow loss of flesh and strength. *Physical signs:* On inspection there are cyanosis; perhaps clubbed fingers; round shoulders; a short, thick neck; the *barrel-shaped chest*, which is constantly in the position of inspiration, moves little in breathing; the sternum is high and prominent; the ribs stiff and wide apart; breathing is labored; the apex-beat hidden; epigastric pulsation and dilated venules are seen along the margin of the ribs. Palpation confirms the above points. Fremitus is diminished. Percussion: Hyper resonance or a wooden note is present, extends one or two spaces below the usual limit of resonance, and obliterates or decreases the area of heart dulness. On auscultation the breath sounds are feeble; inspiration short; expiration very long and low pitched; the voice feeble or increased; there are râles from bronchitis, sibilant and sonorous or moist; and the pulmonary second sound is accentuated.

Prognosis.—The patient runs along for years and finally has symptoms of failure of the heart's compensation, acute pneumonia, lung tuberculosis, or dies of other chronic productive inflammations.

Treatment.—A dry equable climate is best. General hygienic care is needed.

(4) **Atrophic** or **senile emphysema** is a late senile change in withered old persons. There is a small chest with sloping ribs, and the lungs are small.

QUESTIONS.

Give the causes and symptoms of chronic interstitial pneumonia.
What is pnemonoconiosis?
Give the varieties of emphysema.
Give the symptoms of idiopathic emphysema.
Give the treatment of emphysema.

ABSCESS OF THE LUNG.

Etiology.—The bacteria are the pneumococcus, the pyogenic streptococcus, or staphylococci, alone or combined with other forms. They enter (1) by the bronchi in pneumonias, especially tubercular and aspiration pneumonia; with infarcts; inflammations or operations on the upper air-passages. (2) By the blood-vessels, chiefly as septic emboli. (3) By direct invasion from wounds of the chest or lung, from pleurisy, liver abscess, etc.

Lesions.—There may be a single large abscess, a few inches in diameter, or many small abscesses. With recovery scar-tissue forms.

Symptoms.—There are the symptoms of the primary condition. The abscess causes cough at intervals; the sputum is abundant, has a foul, acid smell, contains much yellowish or greenish pus, fragments of lung tissue, bacteria, and detritus. The *physical signs* of a large abscess are those of a cavity surrounded by consolidation. The multiple small abscesses cause irregular signs of consolidation, over different areas, with pleurisy. The primary condition also often gives signs. The general symptoms are those of sepsis. Pleurisy, either fibrinous, empyema, or pyopneumothorax, or pyemia may follow. They usually die of sepsis or a complication. Some cases recover after weeks.

The **treatment** of the large abscess is incision and drainage; the other cases should be treated as sepsis and fetid bronchitis.

GANGRENE OF THE LUNG.

Etiology.—Diseased lung tissue undergoes necrosis usually from streptococcus or staphylococcus infection; this dead tissue putrefies from the action of putrefactive bacteria. The primary condition may be pneumonia of any kind, infarcts, cavities, wounds, or lung compressed by an aneurysm, or a perforation from cancer of the esophagus. Diabetes strongly predisposes.

Lesions.—The gangrene may be (*a*) *diffuse* (lobar pneumonia or a large embolus), with a great mass of greenish-black, putrid matter softening in the center; there is no line of demarcation. Or (*b*) *circumscribed*, a greenish-brown area that softens and forms a cavity with shaggy walls containing a putrid liquid. Around this is a zone of exudative inflammation and another of edema. Intense bronchitis is present. The foci may be single or multiple, are often at the surface, and vary in size. A line of demarcation may form and the area be filled with scar tissue, which contracts later.

The **symptoms** and *physical signs* resemble abscess. The sputum is very abundant, extremely putrid, and settles in three layers: on top is a dirty froth, the second is a liquid layer, and the third, at the bottom, is much granular sediment containing fragments of lung, with elastic tissue, bacteria, pus and blood and detritus. There may be pyopneumothorax or other pleurisy, or hemoptysis, or embolism elsewhere. The patient usually dies in a few days from exhaustion, hemorrhage, or a complication. Sometimes there is a slow recovery.

The **treatment** is surgical when possible. Otherwise it is like that of abscess.

QUESTION.

Give the etiology, lesions, symptoms, complications, prognosis, and treatment of abscess and gangrene of the lung.

NEOPLASMS.

Primary tumors are rather rare. Secondary growths are common with tumors of the breast, alimentary tract, bone or mediastinum. Primary tumors are in one lung and often large; secondary tumors often are many and in both lungs. *Associated lesions:* Pleurisies, pneumonia, abscess, gangrene, carcinomatous lymph-nodes.

Symptoms.—There may be cough, pain in the tumor, and perhaps tenderness over it. Sputum is usually scanty, like thin currant jelly, and may contain fragments of the tumor. There may be symptoms from pressure on a bronchus or the trachea, esophagus or nerves, especially the recurrent laryngeal; the symptoms of pleurisy often with bloody effusion; some fever at times; a leukocytosis; and the cachexia of malignant growths. *Physical signs:* Primary cancer gives the signs of ordinary consolidation or massive consolidation (see p. 68) and the signs of dry pleurisy, or pleurisy with effusion that recurs persistently after tapping. Secondary tumors give less regular signs, over both lungs, of pleurisies, or areas of consolidation.

Prognosis.—Carcinoma is usually fatal within a year.

QUESTION.

Give the symptoms of carcinoma of the lungs.

DISEASES OF THE PLEURA.

PLEURISY.

Etiology.—Sometimes it is apparently *primary* after exposure, due to the pneumococcus or tubercle bacillus. *Secondary cases* are more common. The pneumococcus, streptococcus, *tubercle* bacillus, and, less often, other bacteria are present.

Infection occurs by extension from: (*a*) the lung in pneumonias, infarcts, neoplasms, etc.; from (*b*) the chest wall in fractures or other injuries, or caries of spine; (*c*) through the diaphragm from the liver, etc. It occurs in the infectious diseases.

Lesions.—There are four stages: fibrinous, serofibrinous or serous, empyema, and healing.

(1) **Fibrinous Pleurisy.**—Both visceral and parietal layers are affected. At first congested, the surface becomes lusterless; then covered with flakes or a solid layer of fibrin of any thickness up to ¼ inch or even more, the liquid part of the exudate being absorbed at once. The fibrin contains some leukocytes and often bacteria. The endothelium is swollen and desquamated. The area involved may be of any size. This ends in healing or a liquid effusion.

(2) **Serous or Serofibrinous Pleurisy.**—The exudate becomes more abundant, the fibrinogen is not all precipitated, nor is the liquid absorbed, but collects in the lower part of the pleural cavity. It is an exudate with a variable number of leukocytes, endothelial cells, and flakes of fibrin. The amount varies from a few ounces to three or four quarts. Two or three pints is an ordinary quantity. It is usually on one side only and is seldom encapsulated. With large effusion the lung may be compressed and the neighboring viscera displaced. It goes on to healing or to empyema.

(3) **Empyema.**—The liquid gets more turbid and may become thick pus. Sometimes the effusion is pus from the start. The pleura is often much thickened with masses of fibrin, and later with connective tissue. The lung, with large effusions, is collapsed. The largest effusions are pus, and encapsulation is common. The pus may burrow or rupture into the lung, bronchus, pericardium, mediastinum, vena cava, or through the diaphragm, but usually externally by one or more openings in the anterolateral region of the thorax.

(4) **Process of Healing.**—The fibrin may be absorbed and the pleura return to normal, but this is rare. Usually the fibrin is replaced by connective tissue that leaves permanent adhesions and a thickened pleura. Less often a progressive chronic productive inflammation of the pleura follows. *Serous form:* The serum is finally absorbed, and healing pro-

ceeds as in the fibrinous form. *Empyema:* If healing takes place, the pus-cells degenerate, disintegrate, and, together with the liquid, are absorbed, and healing proceeds as previously mentioned. Or there may be left a cheesy residue that becomes encapsulated with connective tissue and sometimes calcified. The pleura is thicker and the chronic productive pleurisy is more common in empyema than in the other forms and spontaneous healing is rare.

Symptoms.—(1) *Fibrinous or Acute Pleurisy.*—General symptoms are sometimes absent or there may be abrupt onset with chill, fever (101°–103° F.), and leukocytosis. The local symptoms are pain on breathing, coughing, or other movements of the chest, stabbing in character, referred toward the base of the affected lung, usually in the axilla or further forward, and a short dry cough. *Physical signs:* Rapid, shallow, jerky breathing, more marked on the affected side. Over the affected area fremitus is occasionally lessened and there may be tenderness. The percussion note occasionally is a little dull. Breathing and voice may be feebler. There are pleural crepitations, heard mostly at the end of inspiration or with coughing. This is the important sign. *Course:* The fever goes by lysis within a week; the pain lessens, but is apt to go more slowly and to recur at times for a few weeks. Often the primary disease is the chief part of the clinical picture.

(2) *Serous or Serofibrinous Pleurisy.*—This may follow acute fibrinous pleurisy, but often the onset is insidious, and after a week or two the patient realizes that he is ill. Usually there is some fever (100°–102° F.) for a week or more, that disappears slowly, also a gradual loss of flesh and strength, slight leukocytosis, increasing dyspnea on exertion, and cough with little mucous sputum. *The physical signs of the stage:* On inspection, accelerated breathing; restricted motion of the affected side, which is ½ to 1 in. larger than the other, at least in expiration. *Below the level of the liquid* there is on *palpation* no respiratory movement, and fremitus is absent or lessened; on *percussion*, flatness or marked dulness, with increased resistance; on *auscultation*, breathing is absent or feeble or bronchial; voice absent or feeble or bronchial, and often of a nasal quality; there are

no râles. *At the level of the liquid* is a zone of transitional signs. There may be dulness, bronchovesicular breathing, egophony, and, perhaps, friction sounds. This level is not horizontal, but a curve; it changes slowly, and often little with a change of the patient's position. *Above the level of the liquid* the signs are normal or exaggerated, and resonance is often tympanitic. A needle withdraws the serum. As the liquid disappears, by absorption or tapping, the "level-of-the-liquid" signs recede to the bottom of the lung, leaving in their wake some dulness and friction sounds that slowly disappear after weeks. The heart may be displaced away from the effusion, especially with left pleurisy. There is dulness in Traube's semilunar space with left pleurisy. The liver may be pushed down with right pleurisy. Over the other lung there may be exaggerated signs. *Course:* Favorable cases may last only four weeks; others last several months and show little tendency to recovery. Secondary cases or cases in weak persons may be fatal.

(3) *Empyema.*—This is like the former type, but the fever is more severe and of a septic type; loss of flesh and strength, and leukocytosis are marked. The *signs* are the same, but encapsulated pus and large effusions are more common. The needle withdraws pus. The chest wall may be edematous. *Course:* There is little tendency to healing. It may rupture externally with sinuses that discharge for months or years, and amyloid disease may follow; into a bronchus or the lung with expectoration of much pus and perhaps a slow recovery, or pyopneumothorax; into the pericardium or vena cava with death. Death may be due to exhaustion and the *prognosis* of the primary disease is made more serious.

(4) *Special Forms.*—In *diaphragmatic pleurisy* there is an exudate on the diaphragmatic pleura; the pain is epigastric; the breathing costal; there may be tenderness along the tenth rib and cartilage; the signs are negative, but the general symptoms are often severe. *Encapsulated pleurisy:* Serum or pus is walled in by adhesions. The signs are often indefinite. The diagnosis is made by finding the liquid with a needle. In *interlobar pleurisy* the exudate is encysted in a fissure of the lung. In *hemorrhagic pleurisy* the effusion is bloody serum. It occurs with tuberculosis, cancer, or asthenic

conditions. With *double pleurisy* with effusion there is usually pericarditis. *Pulsating pleurisy* is an encapsulated serous or purulent effusion that transmits the heart-beats. Usually it is on the left side.

Treatment.—*Fibrinous.*—Strap the chest with adhesive plaster applied very tightly in full expiration, two-thirds around the chest. Morphin may be needed for pain. *Serous.*—Thoracentesis: The patient is half-reclining. After cleaning the skin, thrust a trocar and canula or aspirating needle into the first space below the angle of the scapula, or into the sixth or seventh space in the mid-axilla. The liquid is allowed to run out or is slowly aspirated. After withdrawing the instrument, seal the wound with sterile gauze and adhesive plaster. With marked dyspnea or very large effusions, tap at once. With smaller effusions, some advise waiting for a week or two. During the tapping there may be great pain in the lung or side, violent coughing, weakness of the heart, syncope, collapse or, very rarely, sudden death, or collapse of the lung and pneumothorax. Rarely tapping may be followed by congestion or edema of the lung or profuse serous albuminous sputum. The amount of liquid withdrawn varies in the practice of different authorities, from a definite maximum (10–30 ounces) to as much as can be removed. If the liquid accumulates, tap again. Other treatment: Instead of tapping or after tapping restrict the liquids ingested and increase the liquid excreted by diaphoresis, purging, and diuresis. Apply counterirritation to the chest. To expand the lung use deep breathing or James's bottles. *Empyema* is surgical. In all forms general hygienic measures and treatment of the general condition are needed. Iodid of iron, cod-liver oil, or other tonics are given.

CHRONIC DRY PLEURISY.

After other pleurisies the pleura is often thick and adherent, the extent depending upon the duration of the pleurisy and the nature of the exudate, being least in fibrinous pleurisy and most in empyema. The connective-tissue formation usually ceases with healing of the original condition, and such cases are not strictly chronic dry pleurisy. Occasionally a real chronic pleurisy follows, or it may be primary. It also forms one type of tuberculous pleurisy.

Lesions.—A chronic productive inflammation of the pleura without exudation. Progressive thickening and adhesions of the pleura are found, and the process extends into the lung as interstitial pneumonia.

Symptoms.—General symptoms usually are absent, except for occasional pain or discomfort in the affected part. *Physical signs:* The affected side of the chest shows restricted movement, defective resonance, fremitus, breathing, and voice; and creaking râles. There are in time the signs of interstitial pneumonia (see p. 223).

The **course, complications,** and **prognosis** are those of interstitial pneumonia.

Treatment.—*Preventive:* Early and careful treatment of the original pleurisy. The later treatment is that of interstitial pneumonia—tonics, hygiene, and a dry, warm, equable climate.

QUESTIONS.

Give the varieties of pleurisy, the symptoms, and course of each kind.

Give the treatment of pleurisy.

PNEUMOTHORAX.

Etiology.—(1) Perforation of the lung and its pleura, usually from a necrotic focus in tuberculosis, less often in abscess, gangrene, or empyema; from sudden muscular effort; a wound from a fractured rib, aspirating needle, etc. (2) Perforation of the outer layer of the pleura, in wounds of the chest wall; perforation from necrotic processes in the esophagus, or through the diaphragm after ulceration of the stomach or colon. (3) Decomposition of pleural exudates may lead to formation of gas without perforation. Pneumothorax occurs more commonly in adults, in males, on the left side; and perhaps emphysema predisposes.

Lesions.—In pneumothorax proper there is only gas in the pleural sac. Almost always an exudate is added, either serous (hydropneumothorax) or pus (pyopneumothorax).

The affected side is larger; the pleural sac is widely distended. At the bottom is the effusion; above is the gas, which is air (except in the putrefactive cases), and it is often at a pressure greater than atmospheric. The lung is collapsed around its root, and often compressed against the spine. The pleura is inflamed. The diaphragm, liver, and spleen are pushed down; the heart and other mediastinal contents are displaced to the other side, usually beyond the middle line. There is the primary causative lesion, usually a tuberculous focus.

Symptoms.—The onset is usually sudden, with sharp pain in the lung at the site of rupture, urgent dyspnea, some cyanosis, and more or less collapse. The temperature at first is often subnormal, but soon rises, and is apt to be of a septic type. *Physical signs:* The cyanosis and dyspnea are seen; the affected side is larger, perhaps by 2 inches; the spaces may bulge, and there is no respiratory movement. The apex-beat is displaced to the other side, perhaps beyond the middle line. Over the liquid at the bottom of the chest are no fremitus, flatness, no breathing or voice or râles. The surface of the liquid is always a horizontal plane, whatever the position of the patient. Over the gas in the upper part of the chest fremitus is feeble or absent; the percussion note ranges from a clear amphoric tympany to marked dulness. Wintrich change of pitch is present (lower with mouth closed, higher with it open). On auscultation, the breathing and voice are amphoric, loud or feeble, or often absent. The following *special signs* are heard best with the ear on the chest at or a little above the surface of the liquid: The "metallic tinkle" sounds like water dropping into liquid in a resonating chamber. Metallic moist râles are present. "Hippocratic succussion" is a splashing sound heard when the patient is shaken. "Coin sound": a coin is pressed against the chest and tapped with another coin; the sound is distinctly heard by the listening ear. Also a needle may release gas or find liquid alternately by varying the position of the patient. Toward the apex of the chest, especially behind, there may be an area of signs of more or less consolidated lung. The signs of marked displacement of the heart and of either the liver or spleen are present. Over the other side of the chest all signs are somewhat exaggerated.

Course.—Air alone may be absorbed in a few days with recovery. The liquid effusions may be slowly absorbed with the same result. There may be attacks of cyanosis with threatening or actual asphyxia. Usually the patients die of exhaustion and sepsis within a few weeks. Some very acute cases succumb in the initial collapse within a few minutes or hours. Weak bedridden consumptives may show no acute symptoms, and the condition is discoverable only by physical signs or autopsy.

Treatment.—Marked cyanosis and threatened asphyxia may often be relieved by letting the gas escape through a fine needle. Effusions should be treated in the usual ways. Besides this, rest in bed and causal and symptomatic treatment are indicated.

QUESTION.

Give the etiology, lesions, symptoms, and treatment of pneumothorax.

HYDROTHORAX.

Etiology.—Usually it is part of general dropsy due to disease of the heart or kidneys. Some cases are due to local causes, such as compression of the vena cava, or cancer of the lung or pleura.

Lesions.—The primary condition and the general dropsy are present. The pleura is not inflamed, but may be edematous. It contains clear serum, free from fibrin; the specific gravity is less than 1015; there are few cells and relatively little albumin. It is a transudate.

Symptoms.—Besides those of the primary condition there are dyspnea, usually developing slowly, and a slight cough, but no pain. The *physical signs* are like serous pleurisy, but it is usually bilateral, and never encapsulated; there are no friction sounds; the aspirated liquid is a transudate.

The **prognosis** is that of the general condition, but sometimes it develops rapidly and kills in a few days.

Treatment.—That of the primary condition and tapping.

QUESTIONS.

Give the etiology, lesions, and symptoms of hydrothorax.
Give the differential diagnosis from pleurisy with effusion.

NEW-GROWTHS.

Primary tumors are very rare. **Secondary** are more common, from tumors of the lung and other neighboring viscera.

Symptoms.—The symptoms of cancer in general and of the primary tumors are present; also those of fibrinous or serous pleurisy. The exudate is apt to contain blood, fatty endothelium, and possibly some tumor cells. It reaccumulates rapidly after tapping.

Treatment.—The liquid may be removed repeatedly by tapping.

QUESTION.

Give the causes and symptoms of new-growths of the pleura.

DISEASES OF THE CIRCULATORY SYSTEM.

THE PERICARDIUM.

PERICARDITIS.

Etiology.—*Bacteria:* Pneumococcus, Streptococcus pyogenes, Staphylococcus aureus, tubercle bacillus, gonococcus, etc., or sometimes none. (*A*) It is usually secondary (1) to rheumatism, but is seen with chorea, pneumonia, scarlatina, and occasionally with any infection; (2) in chronic nephritis, scurvy, and gout; (3) may extend from pneumonia, pleurisy with effusion (especially if bilateral), acute myocarditis, aortic heart disease, or from any neighboring inflammation. (*B*) Primary cases are described. In general it is more common in childhood and old age and in males.

The **lesions** are like the pleurisies: (1) **Acute fibrinous pericarditis**: The pericardium is dull and congested, then covered with fibrin, that may form a thick, shaggy, or honeycombed mass; both contiguous layers are involved over all or a part of the pericardium; it usually begins near the base of the heart; serum does not accumulate; this ends in healing, a serous effusion, or chronic productive inflammation. (2) **Serous or serofibrinous pericarditis**: The exudate changes; serum collects, from a few ounces to three pints; usually it is clear, yellowish, 1015 or higher, contains much albumin, few leukocytes and epithelial cells, and often bacteria and flakes of fibrin; it may be bloody in tuberculosis, cancer, or in the aged; very rarely it is chylous; the pericardium becomes somewhat thickened with connective tissue; in the heart there may be edema, or exudative inflammation in the myocardium; after lasting a variable time the process tends to heal or to become purulent. (3) **Purulent pericarditis**: The liquid becomes purulent or even thick pus; sometimes it seems to be purulent from the start; the serous membrane is much thickened with connective tissue, and its surface is covered with fibrin or granulation tissue; the superficial layer of the heart shows myocarditis and fatty degeneration; it tends to persist, but may heal or rupture. (4) **Process of healing**: The fibrin is absorbed and replaced by connective tissue with adhesion of the two layers of the pericardium. Serum is absorbed sooner or later and healing proceeds as above, but the pericardium is more thickened. With pus, the liquid and the débris from the degenerated cells and the disintegrated masses of fibrin are absorbed, or there is left a cheesy mass which may be encapsulated by connective tissue, and calcified. Adhesions form as above, but there is more thickening of pericardium. Spontaneous healing with pus is not common.

Symptoms.—(1) *Fibrinous Pericarditis.*—There are the symptoms of the primary disease, usually acute articular rheumatism; the pericarditis may give no subjective symptoms or there may be some precordial distress—a sense of constriction or, perhaps, pain near the xiphoid cartilage; it may radiate to the left shoulder, or be sharp and stabbing, and is more common early; there may be dyspnea, palpitation, slight fever, and more prostration. *Physical signs:* On *inspection* a forcible accelerated apex-beat. On *palpation*, perhaps a friction-fremitus, systolic and diastolic or irregular, felt best at the fourth or fifth left spaces near the sternum, with the patient leaning forward. On *auscultation*, the heart sounds are nearly normal, the action forcible and accelerated; there is a pericardial friction sound, rubbing or creaking, close to the ear, systolic or diastolic, but usually double and not exactly synchronous with the cardiac phases; it is loudest at the fourth and fifth left spaces close to the sternum; is not transmitted, is increased by pressure of stethoscope, louder in the erect or procumbent posture, is very inconstant and its site changes often; the pulse is accelerated, regular, and of good tension. In weak patients the pulse may become weak, very rapid, and irregular. A pleuropericardial friction is sometimes heard with pericarditis (and also without it) at the left border of the precordium near the apex; it shows two rhythms, cardiac and respiratory, is louder in expiration and less in inspiration. *Course:* This form lasts a few days or weeks, and is followed by recovery or serous pericarditis. *It does* not kill.

(2) *Serous or Serofibrinous Pericarditis.*—As the serum collects there may be marked dyspnea, often orthopnea; pain increased by pressure on the precordium; fever (102°–103° F.), lasting several days and falling by lysis; and greater prostration, which may be extreme. There may be great nervousness, melancholy, low delirium, stupor, and even coma; pressure symptoms may be added—dysphagia, greater dyspnea, cough, dusky skin, and swollen veins in the neck. *Physical signs:* On *inspection*, the apex-beat is less distinct and may be displaced upward and to the left; often it is invisible; with large effusions there may be bulging of the precordium and lessened expansion of the left side of the chest; the epigastrium may be prominent. *Palpation* finds the apex-beat as above; a friction-fremitus may be felt near the base. *Percussion:* The area of precordial dulness is enlarged in all directions and pyriform or triangular, with its apex at the sternum opposite the third rib or second space; its base is lost in the liver dulness; the right border one or two inches to the right of the middle line in the fifth space; the left border may extend to the anterior axillary line and be traced downward to the sixth space. Beyond this border there may be a zone of tympany over the left lung. There may be dulness in the left interscapular region. If the patient lies down these areas are smaller. *Auscultation:* At the apex the sounds are faint or even absent. The apex when located may be an inch or more within the area of dulness; a systolic blow is common. At the base the pulmonary second sound is accentuated, and all the sounds are louder than at the apex; there may be a friction-sound. Over the tympanitic area there may be bronchovesicular breathing and voice and some fine râles. A needle finds serum. The pulse is accelerated, but of surprisingly good force when compared with the heart sounds. In large effusions it may be weak and irregular or pulsus paradoxus (a radial pulse that shows exaggerated respiratory variation, being larger during expiration and smaller or even not felt during inspiration). During recovery the signs gradually become normal, except that the friction-sound returns or becomes more marked. *Course:* The liquid increases for two days to a week or two, persists for a few days or many

weeks, and may be absorbed rapidly or slowly. The more protracted cases are called chronic pericarditis. Moderate cases recover. Severe attacks or those in weak persons may kill from asthenia in two or three weeks; it is often a terminal event in the aged. The *prognosis* of the primary disease is made more grave.

(3) *Purulent pericarditis* (rare in rheumatism) may follow serous effusion or be purulent from the start. There are the general symptoms of sepsis; the other symptoms and the physical signs are like the serous form, except that large effusions are rather common, there may be edema of chest wall, and the needle finds pus. *Course:* The mildest cases may be followed by slow recovery, but usually the patient dies of sepsis in a few days or weeks. It may rupture internally with speedy death; or through the chest wall with a sinus and chronic sepsis.

Treatment.—The primary disease should be treated. *Fibrinous form:* Cardiac rest is aided by keeping the patient in bed on a scanty diet and putting an ice-bag over the heart. An *effusion* may be treated by a dry diet, purging, diaphoresis, diuresis, blisters over heart, or potassium iodid, about 30 grains a day. If paracentesis is used, the trocar and canula or the needle is inserted (*a*) into the fourth left space one inch from the sternum, or (*b*) the fifth left space one and one-half inches from the sternum, or (*c*) upward and backward in the left costoxiphoid angle, or (*d*) in the fifth right space close to the sternum. For *pus* an incision with resection of rib cartilages and drainage is needed.

QUESTION.

Give the etiology, the varieties of pericarditis, and the lesions, symptoms, prognosis, and treatment of each variety.

ADHERENT PERICARDIUM (Chronic Adhesive Pericarditis).

Etiology.—It is an occasional sequel to the foregoing forms. It is more common in children and is often tubercular.

Lesions.—These cases have a chronic productive inflammation that unites the heart to the outer layer of the pericardium and extends to the mediastinum, uniting the pericardium with the pleura and the front wall of the chest. The heart is much dilated and hypertrophied. There may also be a chronic productive peritonitis, perihepatitis, perisplenitis, and ascites. Simple adhesions between the heart and outer layer of the pericardium usually give no marked symptoms, and are not properly included here.

The **symptoms** are chiefly those of the dilatation and hypertrophy of the heart. *Physical signs:* Besides the signs of a very large heart there are, on *inspection*, bulging of the precordium; the heart impulse is extensive, wavy and diffuse, reaching the sixth space and often the anterior axillary line; there is systolic retraction of the apex region and of the cartilages of the lower left ribs (eleventh and twelfth); also diastolic collapse of the veins of the neck. On *palpation* there is a distinct precordial shock in diastole. The apex-beat does not move when the patient turns on the left side. The pulsus paradoxus is often present.

The **course** is that of the dilatation and hypertrophy.

Hydropericardium.—The *etiology* is that of general dropsy, or there may be local obstruction of the vena cavæ. The *symptoms* and *signs* are those of pericarditis and the primary condition, but the liquid is a transudate, and there is no fever and no friction signs. The *prognosis* is that of the causative condition.

Hemopericardium follows rupture into the pericardium of aneurysm of the aorta, the heart, or coronaries, rupture of the heart or wounds of the heart or pericardium. The pericardium is distended with blood. There are sudden dyspnea, collapse, perhaps cyanosis, or marked pallor, very rapid feeble heart action and the signs of liquid in the pericardium. The patients die after a few seconds or a day or two.

Pneumopericardium follows wounds of the pericardium and chest wall; rupture of the lung, usually a tuberculous focus, or of the esophagus or stomach. Very rarely gas is produced by the Bacillus aërogenes capsulatus in a pericardial effusion. There is gas in the pericardium, usually air. There is also inflammation of the pericardium with an effusion usually purulent. The *symptoms* are those of a very acute pericarditis with effusion of pus. *Physical signs:* Over an area like that of pericarditis with effusion there are, with the patient erect, an upper space of marked tympany, and a lower zone of flatness. The sounds of the heart are absent or feeble. There are splashing or churning sounds of metallic quality. Friction-sounds are usually absent. With the patient lying down there is tympany over all the precordium. The patients die in a day or two. *Treatment* is that of pericarditis with effusion of pus.

QUESTIONS.

Give the etiology, lesions, and symptoms of adherent pericardium.
Give the etiology and symptoms of hydropericardium.
Give the differential diagnosis from pericarditis with effusion.
What is hemopericardium?
What is pneumopericardium?

DISEASES OF THE HEART.

ACUTE SIMPLE ENDOCARDITIS.

Etiology.—Bacteria usually are demonstrable; the Staphylococcus aureus, pneumococcus, Streptococcus pyogenes, or, less often, B. coli communis, gonococcus, B. diphtheria, typhoid bacillus, etc. It is usually secondary to acute rheumatism; sometimes to scarlatina, pneumonia, or tuberculosis of the lung; and rarely to any infectious disease, chorea, chronic gout, nephritis, diabetes, cancer, or other states of malnutrition. It may occur in chronic endocarditis (recurring endocarditis); is more common in children, but occurs in adults also and even in the fetus.

Lesions.—It is acute productive inflammation with formation of *vegetations.* These are nodules up to ¼ inch in diameter. *Microscopically,* in the nodule are a mass of small round cells (proliferated connective-tissue cells), proliferated and polyhedral endothelial cells, and on the surface some fibrin enclosing leukocytes, blood-plates, and bacteria. Any of these factors may predominate. Usually on the auricular surface of the mitral valves, but often on the ventricular surface of the aortic valves, they are arranged in a row along the inner edge of the surface of contact with the other flaps; sometimes on the chordæ tendineæ, or on the ventricle wall. They may slightly constrict the orifice or make the valve incompetent. In the fetus the lesion is on the tricuspid or pulmonary valves. The fibrin may be loosened and form emboli. There may be superficial ulceration. In recurring endocarditis these fresh lesions are associated with the lesions of chronic endocarditis. *Termination:* The connective tissue shrinks, but leaves the valve either functionally normal or somewhat impaired. Usually the productive inflammation persists, becoming chronic endocarditis. Ulceration occasionally becomes marked and merges into malignant endocarditis.

Symptoms.—The active or convalescent primary disease is present. The endocarditis often causes no noticeable symptoms; or there may be some acceleration or irregularity of the pulse, palpitation, precordial distress or even pain, an increase in fever and leukocytosis. The *physical signs* are not many. The apex-beat is seen and felt to be more forcible, accelerated, perhaps irregular; later it may be weaker. A systolic thrill at the apex is rare. *Auscultation:* At the apex the first sound may be prolonged and rough; after a few days there may be a blowing systolic murmur scarcely transmitted. At the base there may be a systolic murmur over the aortic or pulmonary area, not transmitted; or an accentuated or reduplicated second sound. In recurring endocarditis the pre-existing signs may be changed.

Course.—The fever falls by lysis after one to a few weeks. Embolism is rare. The signs may clear up and the heart remain functionally normal. Usually the symptoms of chronic endocarditis develop either at once or after an interval of months or years.

The **diagnosis** is hard to make because murmurs may be present without valve lesions.

Prognosis.—It is not fatal in itself, but there is danger of chronic endocarditis.

Treatment.—If endocarditis is suspected the patient should rest in bed for several weeks and activity must be very slowly resumed. *Symptomatic treatment* may be indicated.

MALIGNANT ENDOCARDITIS.

(Ulcerative, Septic, or Mycotic Endocarditis.)

Etiology.—The bacteria are the same as in the simple form. Primary cases may occur, but usually it is secondary to the same diseases as the simple form; but is rare in rheumatism, and common in pneumonia, sepsis, and pyemia; occurs in gonorrhea, meningitis, rarely in any other infectious disease; also in chronic nephritis or endocarditis.

Lesions.—Like the simple, but the bacteria are constant and more abundant; ulceration is the rule, is more extensive, and may lead to localized bulging or perforation of a valve, may sever a chorda tendinea or erode the heart wall. Embolism with embolic abscesses is common. *Mural endocarditis* is less rare, and occurs on the upper part of the interventricular septum or on the left auricular wall. The process may extend into the pulmonary artery or aorta. There is also the lesion of the primary disease or focus.

Symptoms.—Cases usually are divided into groups; the most important are pyemic, typhoid, cardiac, and meningeal. (1) The **pyemic group** usually has some septic focus. There is a typical pyemia (see p. 76) with embolic abscesses in the brain, spleen, kidneys, lungs, intestines, etc.; and sometimes cardiac pain and dyspnea. The *physical signs* are like the acute simple form, but the pulse and heart action become weaker, faster, and irregular; and the murmur is apt to be rather loud. (2) **Typhoid cases** are common. They are less regular than typhoid; the fever is apt to begin abruptly with chills and to be more remittent; sweats are common; prostration is marked in three or four days; the rash is irregular in time of appearance, duration, and site; it is often petechial; it may be absent; the specific typhoid reactions and complications are absent; there is leukocytosis; the causative bacteria may be found in the blood; there are the heart symptoms and often the embolism. The disease is apt to change to the pyemic type. It is regularly fatal. (3) **Meningeal cases** are clinically like meningitis, but in time are apt to develop pyemia and embolism and heart symptoms. (4) **Cardiac cases**: A large group, called *recurring malignant endocarditis.* There is a secondary infection of diseased valves. In addition to the history and symptoms of chronic endocarditis there are fever (103°–104° F.), often with chills and sweats, bacteria in the blood, leukocytosis, septic embolism, and changing heart signs, perhaps with new murmurs.

Prognosis.—Regularly fatal; the most acute cases last a few days; the usual cases from two to four weeks; often the pyemic cases last into the second month and sometimes into the fourth; the cardiac cases are apt to last even longer and are sometimes called subacute. Occasionally borderline cases between this and simple endocarditis improve and become chronic.

Treatment.—As in pyemia. Antisera may be used if appropriate for the bacteria present.

QUESTIONS.

Give the etiology, lesions, and symptoms of acute simple endocarditis.

What are the prognosis, terminations, and treatment?

Give the etiology and the lesions of malignant endocarditis.

Give the clinical varieties and the symptoms of each type.

Give the prognosis and treatment.

Give the differential diagnosis between ordinary pyemia and the pyemic form; between typhoid fever and the typhoid form.

CHRONIC ENDOCARDITIS.

Etiology.—It may follow acute endocarditis, or be chronic from the start. It may follow alcoholism, syphilis, persistent, excessive muscular exertion or gout. It is associated in causation with the other chronic productive inflammations.

Lesions.—A chronic productive inflammation involving the valves especially, which become permanently deformed. They become thicker, stiff, rough, shortened, adherent along the edges, sometimes calcified, and sometimes they have thrombi on the roughened swellings. As a result, the flaps do not close the orifices perfectly and allow leaking; this is "insufficiency" or "incompetence," and allows of "regurgitation." Or the orifice may be partly occluded; this is "stenosis." The two conditions often coexist, in fact, stenosis seldom or never exists without some insufficiency. The orifice may be reduced to a mere slit, or a very small hole, perhaps only ¼ inch in diameter, at the end of a funnel-like process formed by the irregular adherent cusps. The process may involve the tendinous cords, resulting in shortening and thickening; or may be found on the mural endocarditium. There are subsequent changes in the muscle of greatest importance clinically; hypertrophy of the walls, dilatation of the cavities, degeneration of the muscle-fibers, and fibrosis. There is often chronic productive inflammation of the arteries and kidneys, and emphysema.

QUESTION.

Give the causes and lesions of chronic endocarditis.

CONCERNING COMPENSATION.

A heart that is equal to the work demanded of it is said to be compensated. A heart not equal to this is said to be uncompensated. The ability to keep up the circulation so that every organ receives enough blood depends upon an equilibrium among the mechanical factors entering into the circulation—*i. e.*, the arteries, valves, and force of the heart-pump. Normally the heart is able to do more work than it is ordinarily required to do. Some hearts can do a great excess of work, others a less excess, but in all normal persons this reserve capacity is considerable. Furthermore, by carefully exercising the heart muscle (training) this reserve capacity can be much increased. A limit to this increase exists: thus, no one has yet trained his heart to allow of his running a mile at a hundred-yard pace.

Changes in the arteries or a defect in the valves demands increased force of the heart to maintain the circulation properly. These changes are slow, and the heart automatically adjusts itself to the new conditions as they develop, increasing its force and the amount of work that it does, so that an amount of oxygenated blood is supplied to all of the capillaries that is adequate for ordinary activity, and the "heart is compensated," although when such a person is walking moderately on a level surface he may need as much heart energy as a normal person does when running or climbing a mountain. Indeed, this hypertrophy may be so great that a considerable reserve force is developed, and even under the added disadvantages such a person may even do hard work.

So long as this compensation is maintained the person may not suspect that he has a disordered heart or is not in perfect health. Such hearts are often discovered in routine examinations. Sooner or later, however, the excessive demands made upon the heart tell upon it, and, as the overworked muscle gives out, the blood supply to the organs ceases to be adequate for any unusual exertion, such as climbing stairs or running. Later even ordinary activity is associated with distress or becomes impossible. Again, from disease of the heart muscle, even without significant lesion of valve or artery, the same insufficiency of heart force may develop. In any of these conditions there is said to be "disturbance of compensation." With proper treatment under favorable circumstances, the equilibrium of the circulation may be again restored for a longer or shorter time—"compensation is re-established." Finally, compensation is apt to be "permanently lost" beyond the powers of recuperation.

Whether the primary change is in the arteries, valves, or heart muscle, the symptoms of lack of compensation are pretty much the same. There may be a few symptoms that suggest the special starting-point of the trouble, but the student should first know the general symptoms and it will then be an easier matter to learn the special symptoms of the various lesions.

These general symptoms are largely those of venous congestion: Cyanosis of lips and extremities, perhaps clubbed fingers, distended veins; gastric pain and indigestion, vomiting, hematemesis, constipation, or attacks of diarrhea, hemorrhoids, blood in the stools, enlargement and some tenderness of the liver, occasionally slight jaundice; a large spleen; nose-bleed, dyspnea on exertion, constant dyspnea, or orthopnea; irregular breathing, chronic bronchitis, hemoptysis, pneumonia of heart disease (an interstitial pneumonia with brown sputum); vertigo, headache, disturbed sleep, perhaps delirium, stupor, convulsions. The urine is less, is of high specific gravity, with little or no albumin, and perhaps a few hyaline or granular casts; urea is nearly normal. Later there may be chronic nephritis. Dropsy appearing at first in the ankles at evening and disappearing during the night; later it extends upward and may not wholly disappear during the night; it may become general, involving all of the serous sacs, the connective tissue everywhere, and the lungs. There are loss of flesh and strength. The heart symptoms depend somewhat on the primary lesions, but in general there is more dilatation with its physical signs (see p. 251); there may be palpitation or cardiac distress or pain of any degree, even angina. The first symptom may be dyspnea on exertion, or some palpitation or cardiac distress, or gastric symptoms, or dropsy. Finally, others are added and the patient becomes bedridden. They die feeble and emaciated, or of dyspnea, dropsy, cerebral symptoms, suddenly of heart-failure, or of some complicating or intercurrent disease.

Treatment.—Rest in bed; a light diet, chiefly milk. Sometimes they may need digitalis, about 10 gr. of the leaves, or 1 dr. of the tincture, or 2 oz. of the infusion daily for a few days; continued in smaller doses for one to several weeks. Palpitation may call for an ice-bag over the heart. Stimulants may be needed—strychnin, camphor, etc. For the dropsy—paracentesis, diuretics, salicylate of sodium and theobromin, etc., saline purges. With very marked congestion, cyanosis, and weakening pulmonary second sound, blood-letting (10–20 oz.) may save life.

As compensation returns, the patient may resume activity very gradually, and may resort to carefully graduated exercise. The causative condition may be amenable to treatment.

QUESTIONS.

Discuss compensation.
Give the symptoms and treatment of failure of compensation.

HYPERTROPHY AND DILATATION.

HYPERTROPHY.

Etiology.—Hypertrophy follows increase of pressure in the vessels or other mechanical interference with the heart-action, if at the same time the state of nutrition is good. (1) *Hypertrophy* of the left ventricle alone or of the whole heart is caused by: (*a*) Heart conditions—aortic insufficiency or stenosis, mitral insufficiency, fibrous myocarditis, adherent pericardium, long-continued over-action of the heart, as in exophthalmic goiter. (*b*) Vascular conditions resulting in high pressure—arteriosclerosis, long-continued arterial contraction, or excessive muscular exertion, congenitally narrow aorta, excessive beer drinking. Chronic nephritis is often associated with the arterial disease or spasm. (2) Hypertrophy of the right ventricle is found with (*a*) mitral stenosis or insufficiency, pulmonary insufficiency or stenosis, tricuspid insufficiency. (*b*) Lung conditions—emphysema, or interstitial pneumonia. (3) Hypertrophy of the left auricle, with mitral stenosis or insufficiency. (4) Hypertrophy of the right auricle with tricuspid stenosis or insufficiency.

Lesions.—In **simple hypertrophy** the size of the cavity is unchanged, the wall is thicker. In **eccentric hypertrophy** or hypertrophy with dilatation the cavity is dilated and the wall thickened. This is the usual form. The heart often weighs 20 oz., and, in extreme cases with all parts of the heart involved, 30 to 40 oz. The apex is rounded, the length and width of the heart are much increased, the muscle is dark red, firmer, and tough, especially in the right ventricle. The thickness, measured after rigor mortis has passed off, may be in the left ventricle 1 in. or more; in the right ventricle ½ to ¾ in. The papillary muscles are large and flattened, the septum is thickened, especially with right hypertrophy. In hypertrophy of the left ventricle chiefly, the heart is very long and the left ventricle shows much more than normally from the front. In right hypertrophy the heart is broad, and the right ventricle seems to quite reach the apex. It may be as thick-walled as the left. Hypertrophied auricles may have walls twice as thick as normal. *Microscopically*, the lesion is an increase in the number of muscle-fibers. There may be an increase in the amount of connective tissue also. There are

[1] The normal heart weighs 8 to 10 oz. in the female, and 10 to 12 oz. in the male. It is about 5 in. long and 3½ in. wide. The right auricle has a capacity of about 2 oz., its wall is about 1/12 in. thick. The left auricle holds a little less, its wall is about ⅛ in. thick. The ventricles hold about 3 oz. each. The wall of the right ventricle is about ⅕ in. thick, of the left, about ⅗ in.

the associated lesions in the valves, arteries, lungs, kidneys, etc.

Symptoms.—Hypertrophy is a beneficial process that tends to prevent the development of symptoms. Sometimes, however, it seems to cause headache, tinnitus, flushing of the face; flashes of light; cardiac discomfort or pain that may even suggest angina. In time, arteriosclerosis is apt to develop, with its sequels. *Physical signs.*—**Hypertrophy of the left ventricle**: The heart impulse is seen and felt in the fifth, sixth, or seventh space from 4 to 6 in. to the left of the middle line, it is heaving and forcible; there may be some bulging of the precordium and some widening of the rib spaces. *Percussion:* The area of cardiac dulness is enlarged, extends upward to the third rib or second space; its left border is from 4 to 6 in. to the left of the middle line and can be traced farther downward than is normal; the right border is not much changed. *Auscultation:* At the apex, the first sound is long, loud, booming, and perhaps reduplicated. At the base the aortic second sound is accentuated. The pulse is full, perhaps accelerated, and the tension is usually high. **Hypertrophy of right ventricle**: The heart impulse is seen and felt nearer the sternum; the apex-beat may be displaced to the left (4 to 5 in. from the middle line) and slightly downward (fifth or sixth space). There may be bulging of the gladiolus. *Percussion:* The area of dulness is enlarged, chiefly to the right (1 to 2 in. to the right of the middle line in the fourth and fifth spaces), and also to the left (1 or 2 in. beyond normal). *Auscultation:* The first sound is loudest to the right of the apex, sometimes at the sternum; it is long, booming, may possibly be reduplicated. The pulmonary second sound is accentuated. The pulse is not large or hard; it is accelerated. With **hypertrophy of both ventricles** these signs are combined. They are modified by the signs of the primary conditions in the valves or elsewhere.

DILATATION.

Etiology.—The causes of dilatation associated with hypertrophy have been given. Usually a time is reached when *the nutrition* of the heart muscle fails, the hypertrophy ceases, and the dilatation becomes the striking factor. With little or no hypertrophy and without lesions of the valves or arteries, dilatation occurs in states of poor nutrition, such as protracted fever, anemias, leukemia, and cachexias.

There is an acute form that follows a severe and unusual exertion, such as mountain climbing; or rarely comes on without discoverable cause.

Lesions.—The heart is large, less conical, flabby; the wall may be thinner or thicker (from associated hypertrophy); one or more parts may be involved, but probably the right ventricle is most commonly dilated. The dilated ventricle may hold 6 to 8 oz. or more; a dilated auricle may hold 10 oz. or more. The circumference of the mitral ring may be increased from 3½ in. (the normal) to 5 in. or more; the tricuspid from 4½ in. (the normal) to 6 in.; the aortic (normally 3⅛ in.) and the pulmonary (normally 3½ in.) may be increased an inch. This results in a relative insufficiency of the valves, with its consequences. The heart muscle may be degenerated or fatty, or the seat of interstitial inflammation.

Symptoms.—The **acute form** is rather rare. There is a sudden onset of dyspnea, palpitation, and perhaps cardiac pain. There are marked prostration and the physical signs.

Chronic cases are common. The *symptoms* are those of failure of compensation (see p. 247) and the *physical signs.* **Dilated Left Ventricle.**—*Inspection:* The apex-beat is diffused, wavy, may extend 4 to 6 in. or more to the left of the middle line, and downward to the fifth, sixth, seventh or even eighth space. Often it is more easily seen than felt. *Percussion:* The area of dulness extends upward to the second space; 4 to 6 in. to the left of the middle line; and downward to the sixth or even the eighth rib. *Auscultation:* At the apex the first sound is short, of higher pitch, and more like the second, with a longer first pause (embryocardia). There may be reduplicated sounds or gallop rhythm. There is often a systolic murmur, transmitted to the left. At the base the aortic second sound is weak; the pulmonary louder. The action is weak, rapid, and irregular. The pulse is small, soft, rapid, irregular. **Dilatation of Right Ventricle.**—*Inspection:* The veins may be turgid and often pulsate, the heart impulse is wavy and diffuse near the sternum; epigastric pulsation is common; the apex may not be distinctly made out; the impulse is better seen than felt; it is displaced to the left and a little downward. *Percussion:* Dulness extends farther to the right, 1 to 2 in. to the right of the middle line; also an inch or more to the left. *Auscultation:* The first sound is as given above; there may be a systolic murmur over the lower part of the sternum transmitted to the right. The second sounds may be weak. The action is weak, irregular, accelerated. The pulse as above. Preëxisting murmurs may be modified or lost, and there may be new murmurs due to relative insufficiency of the valves.

The **course** of dilatation and of hypertrophy depends upon the extent of damage to the circulatory organs, the cause, the nutrition of the patient, and the treatment. With good nutrition and proper treatment the compensation often may be restored for a long time. Sooner or later, however, the patient is apt to die from loss of compensation.

Treatment is for failure of compensation and the causes.

QUESTIONS.

Give the causes and lesions of hypertrophy of the left ventricle; also of the right ventricle.

Give the symptoms and physical signs of each.

Give the causes, the lesions, and the symptoms of dilatation of both ventricles.

Discuss acute dilatation.

VALVULAR DISEASE OF THE HEART.

AORTIC INSUFFICIENCY.

Etiology.—(1) Arteriosclerotic cases are most common, and are due to continued excessive work (soldiers, athletes, laborers); chronic alcoholism; syphilis or gout. These are conditions that lead to prolonged high blood-pressure. (2) The endocarditis group follows rheumatism or other infection at once or after an interval. (3) Rupture of a valve (rare) follows great muscular strain with a healthy valve, or less strain with a diseased valve. (4) Congenital cases (not common): the cusps are adherent and often sclerosed. (5) *Relative insufficiency* follows dilatation of the aorta with sclerosis or aneurysm. It is not very rare. In general, while aortic insufficiency is found in the young, it is most common in middle adult life or later, and in males.

Lesions.—The Aortic Valve.—In the endocarditic group the cusps are nodular, calcified, and stiff. In the sclerotic group they are shortened by curling of the edges, but smooth. In relative insufficiency they may be somewhat sclerosed, but the ring is too big for them to close it. In all cases the valve fails to close the orifice, the aorta is often dilated and sclerosed, and coronary sclerosis is common. The reflux of blood in diastole leads to hypertrophy and dilatation of the left ventricle, which become extreme. Later, there may be relative insufficiency of the mitral valve with its effects. This gives the largest hearts (cor bovinum). Still later the dilatation predominates and the muscle degenerates. Some narrowing of the aortic orifice may be associated, but it usually is not significant.

Symptoms.—During the period of fair compensation there may be head-symptoms—headache, vertigo, syncope, flashes of light, disturbed sleep, perhaps melancholy or insanity; also pallor and anemia; cardiac palpation and pain, or angina pectoris; or sudden death. When compensation fails (see p. 247) the above symptoms are more marked, there may be embolism of the spleen, kidney, and brain; and nose-bleed. Anasarca and cyanosis appear very late, if at all. **Physical Signs During Compensation**—*Inspection:* The precordium may bulge in a child; the impulse is heaving, extends much to the left and downward; the apex-beat may be in the sixth, seventh or eighth space, and 4 to 6 in. to the left of the middle line. *Palpation:* The impulse and apex-beat as above. Sometimes a diastolic thrill is felt at the aortic area or over the sternum opposite the third rib or third space. *Percussion:* The area of cardiac dulness is increased, chiefly to the left (4 to 6 in. from the middle line), and downward, (sixth to eighth rib or space), also upward to the second space, and perhaps a little to the right also. *Auscultation:* At the apex the first sound is long and loud, the second often distinct. At the base, the second aortic sound is modified or replaced by a murmur, long, blowing, sometimes musical, loud or faint; loudest at the sternum opposite the third rib;

heard also at the second right space, transmitted down the left edge of the sternum; sometimes heard at the apex Sometimes there is a blowing systolic murmur at the base, transmitted toward the neck at the right of the sternum. Rarely, there is a rolling presystolic murmur at the apex, not transmitted (Flint's murmur). The heart action is regular, very forcible, and not much accelerated. *The vessels:* Visible pulsation of the larger and often of the smaller arteries, and the capillary pulse are present; over the radial or similar arteries there may be a systolic bruit; over the femorals a double murmur upon pressure with the stethoscope. The pulse is very large, fills suddenly, and falls suddenly (water-hammer pulse); the tension varies with the state of the arteries; usually the pulse is regular and perhaps slightly accelerated.

With Failure of Compensation.—The changes in the signs are: The impulse is less heaving and may be wavy; the apex-beat is a little farther to the left; dulness extends more to the left and to the right; the first sound is less loud and is higher in pitch; often there are a systolic murmur at the apex transmitted to the left, and an accentuated pulmonary second sound; the action is weaker, fast, and often irregular; the diastolic murmur may be fainter, the pulse less strikingly water-hammer, fast, and irregular.

Course.—Compensation is often excellent for years and the patient may do very hard work and think himself healthy. When compensation once fails the heart is apt to do badly. Cardiac pain, angina pectoris, and sudden death are more common than in any other valve-lesion. There is apt to be recurring endocarditis with fever for several weeks before death.

AORTIC STENOSIS.

Etiology.—Usually this is associated with general arteriosclerosis, and therefore is most common in older men. Rarely, it is found in younger persons.

Lesions.—The aortic valve segments are thick, stiff, and calcareous; the orifice may be only ½ in. or less in diameter; there is usually some insufficiency also. Rarely, the cusps *are thin,* but adherent along the edges. The left ventricle hypertrophies considerably and later dilates. Still later there may be more dilatation, with relative insufficiency of the mitral and its effects. When the associated aortic insufficiency is marked enough it has its characteristic effect also on the heart. The arteries are sclerosed.

Symptoms.—During good compensation there may be some anemia, dizziness, or headache. When compensation fails there are the usual symptoms (p. 247). **Physical Signs During Compensation**—*Inspection:* The impulse is slow and heaving; the apex-beat displaced downward and to the left; sometimes it can not be seen. *Palpation* verifies the above. A systolic thrill is often felt at the aortic area or over the sternum opposite the third ribs. *Percussion:* The cardiac dulness extends more to the left, 4 to 5 in. from the middle line, and downward to the sixth or seventh space. *Auscultation:* At the apex the first sound is long and loud; the second is distinctly heard. At the base there is a rough blowing systolic murmur, loudest at the aortic area, or the sternum opposite the third space or rib, transmitted upward toward the neck on the right of the sternum. With and after the second aortic sound there may be a faint diastolic murmur. The action is forcible, slow, and regular; the pulse not large, fills slowly, falls slowly, and is not accelerated; the tension varies; the arteries usually are thickened. **With Failed Compensation**—The changes in the signs are a less forcible and more diffused impulse; the apex-beat is less easily made out; when present it is more displaced to the left and downward; the area of dulness extends farther to the left and also to the right; the action is weak, irregular, and accelerated; the sounds weaker and shorter, and the pulmonary second accentuated; the aortic systolic murmur is less loud; often there is a blowing systolic murmur at the apex transmitted to the left. The pulse is very irregular and small, and many heart-beats are not transmitted to the wrist.

Course.—The valve lesion is not suspected for a long time, compensation being very good. Finally, the patient dies of failure of compensation or some other effect of arteriosclerosis, or an intercurrent disease.

MITRAL REGURGITATION.

Etiology.—This is the most common valve lesion. Many cases are due to endocarditis, acute or chronic, following rheumatism or other infection. Many others have relative insufficiency due to dilatation (see p. 250). It is most common in males, and before middle life, but it is not rare at any age or in women.

Lesions.—*With Endocarditis.*—The mitral cusps are thick, short, curled, often calcified, and sometimes adherent at the angles. Without much change in the valve the chordæ tendineæ may be thick and shortened. Either condition may be associated with some stenosis. *In relative insufficiency:* With little or no sclerosis of the cusps the ring may be dilated or the papillary muscles weak. In all cases there is imperfect closure of the valve. Under favorable circumstances there is as a result hypertrophy and dilatation of the left auricle and ventricle, and later of the right ventricle. Finally the dilatation continues and predominates, there is relative insufficiency of the tricuspid valve, and dilatation of the right auricle. With poor nutrition there is dilatation of these cavities with little hypertrophy. The heart is broad. The lesions of venous congestion in lungs and elsewhere are marked.

Symptoms.—**While compensation continues** there may be some cyanosis (and in the young, clubbed fingers), some dyspnea on exertion, a tendency to bronchitis and hemoptysis, perhaps palpitation on exertion. **When compensation fails** it presents the typical picture (see p. 247). An early symptom often is "sleep-start," the patient when falling asleep is startled by a sensation of the heart stopping. **Physical Signs During Compensation.**—*Inspection:* The impulse is heaving and extends from the sternum to the apex-beat, which is displaced to the left, 4 to 6 in. or more from the middle line, and somewhat downward, to the fifth or sixth space. *Palpation* finds the impulse as above, and sometimes a systolic thrill at the apex. *Percussion:* The area of dulness is broadened, extending to the left 4 to 6 in. from the middle line, down to the sixth space, and also to the right 1 to 2 in. from the middle line. *Auscultation:* At the apex the first sound is modified or replaced by a blowing systolic murmur, loud or soft, loudest at the apex, or nearer the sternum, transmitted to the left and heard at the angle of the left scapula; the second sound may be masked by the prolonged murmur or it may be distinct. At the base the pulmonary second is much accentuated. The heart action is of good force, often regular, and usually somewhat accelerated. The pulse is of fair size, not hard, often regular and somewhat accelerated. **When compensation has failed**, the signs change as follows: The impulse is less forcible, wavy, extends more to the left; often is better seen than felt; the thrill is less marked or absent; the heart action is very rapid and irregular in force and rhythm; the murmur at the apex may be heard with only the strongest beats, the pulmonary second sound is at first more accentuated, but later it grows weaker; the pulse is small, soft, very rapid and irregular. The signs of tricuspid regurgitation (p. 259) are often added. The signs of venous congestion are marked.

Course.—Compensation is often quite satisfactory for years. When lost it may again be restored many times, but some irregularity of the heart and pulse are apt to persist. Finally, the patient dies with the symptoms of cardiac failure and anasarca or of some lung complication.

MITRAL STENOSIS.

Etiology.—This is the most common form after acute rheumatism. Sometimes it may be congenital or follow anemia. It is more common in childhood and young adults and in females.

Lesions.—The mitral cusps are thick, short, often calcified, their edges adherent, the orifice constricted, sometimes to a mere slit like a button-hole. The tendinous cords are shortened or quite obliterated, preventing the falling back of the valve. This constricts the orifice and almost always renders it more or less insufficient. A few cases, sometimes called congenital, have cusps grown together like a funnel with an orifice that may be only ¼ inch in diameter, and the cusps are not thickened. The left auricle dilates and hypertrophies. The right ventricle becomes much hypertrophied (often ½ in. thick) and dilated. It seems to reach the very apex. The left ventricle may seem thinner-walled than normal. Later there may be tricuspid insufficiency and its sequels. The mitral regurgitation is often slight; if, however, it is considerable, it produces its usual effects on the heart.

Symptoms.—The symptoms are very like those of mitral regurgitation, but anasarca is less common. **Physical Signs During Compensation**—*Inspection:* There is some bulging of the lower sternal region in children. The impulse is marked near the sternum, the apex-beat may be at the normal site or displaced to the left. *Palpation* confirms the above points, and there is often a presystolic thrill at or just internal to the apex, and it is immediately followed by the apex shock. *Percussion:* The cardiac dulness is increased to the *right*, 1½–2½ in. from the mid-line, also somewhat to the left and upward. *Auscultation:* At the apex or just internal to it is a loud, prolonged, rumbling presystolic murmur, not transmitted, ending in the short, high-pitched first sound. There may be a softer, blowing systolic murmur, transmitted to the left, modifying the first sound. The second sound is sometimes distinct, often feeble or not heard. At the base, the second sound is often reduplicated; the pulmonary second is much accentuated. The action is forcible, regular or irregular, and somewhat accelerated. The pulse varies like the heart action, but is not large or hard. **With failed compensation**: The changes are a wavy impulse, usually no thrill, dulness farther to the right, and also to the left; action very rapid and irregular; the presystolic murmur often absent, the systolic heard with occasional beats; second sound at the base more often reduplicated; the pulmonary accentuated until the heart is very weak, and then fainter. The radial pulse is very small, irregular, and gives no idea of the number of heart-beats. There is often tricuspid regurgitation with its symptoms.

Course.—Compensation may be very good for years; when disturbed it can often be restored many times, but the irregularity of the pulse is apt to persist. There is a tendency to recurring attacks of endocarditis and to embolism, especially in the brain.

TRICUSPID REGURGITATION.

Etiology.—Marked endocarditis is rare, but may occur in the fetus. Relative insufficiency is common from dilatation of the right ventricle.

Lesions.—In relative insufficiency the tricuspid cusps are often slightly sclerosed; the ring and right ventricle are dilated. As a sequel there is dilatation of the right auricle. The degree of hypertrophy of the ventricle and auricle varies greatly.

Symptoms.—Since this is the result of failure of the heart, compensation is not common. At times as compensation is restored the tricuspid insufficiency disappears. The symptoms are those of general venous congestion. The **physical signs with poor compensation** are: *Inspection:* The impulse is wavy, most marked near the sternum, and may be seen to the right of the sternum. Often it is better seen than felt. *Percussion:* Dulness extends to the right, 1½ to 2½ in. from the midline. *Auscultation:* There is a systolic murmur loudest at the lower part of the sternum and transmitted to the right; the sounds are weak. The veins are distended. Systolic pulsation of the veins of the neck, pulsating liver, and cyanosis are present. There are also the signs of the associated lesions.

TRICUSPID STENOSIS.

This is very rare; either congenital or secondary to endocarditis of the left heart. *Physical signs:* At the lower part of the sternum are a presystolic thrill and murmur. Cyanosis is marked.

PULMONARY LESIONS.

Stenosis, usually congenital, is rare; it is due to adhesions of the cusps. Over the pulmonary area there are a systolic thrill and murmur, and a weak second sound. The signs of an enlarged right ventricle are present.

Insufficiency is very rare. It may be congenital or occur in malignant endocarditis. There are a diastolic murmur at the pulmonary area, and the signs of a dilated and hypertrophied right ventricle. The pulse is not water-hammer.

GENERAL PROGNOSIS AND TREATMENT OF VALVULAR DISEASE.

The **prognosis** is worse in children under ten years; better in women than men; varies with the particular valve lesions as given above. With lesion of more than one valve (combined lesions), it is worse.

Treatment.—(1) *Preventive:* In acute endocarditis prolonged rest in bed is the best measure to establish compensation and stop the progress of the disease. Avoid the causes of arteriosclerosis. (2) *During compensation:* Eliminate alcohol, tobacco, tea, coffee. Use moderation in eating, and moderate muscular work—neither too much nor too little. (3) *During disturbance of compensation*, see p. 248.

QUESTIONS.

Give the special causes, the lesions, the symptoms, the prognosis, and the treatment of aortic regurgitation.

Give the same points of aortic stenosis.

Give the same points of mitral regurgitation.

Give the same points of mitral stenosis.

Give the same points of tricuspid regurgitation.

Give the general prognosis of combined valvular disease.

Give the physical signs of combined aortic and mitral regurgitation.

Give the treatment of chronic valvular disease.

THE CORONARY ARTERIES AND MYOCARDIUM.

THE CORONARY ARTERIES.

(1) **Sclerosis** may be a part of general arteriosclerosis or a localized process with dilatation and hypertrophy of the heart. It is an obliterating arteritis. The anterior coronary is more often affected. It leads to fibrous myocarditis, usually near the apex of the left ventricle and in the septum; and to thrombosis. (2) **Thrombosis and Embolism.**—The thrombus forms in a branch of a sclerosed artery. The embolus comes from the heart, the beginning of the coronary, or rarely elsewhere. These lead to *anemic infarct* or *necrosis.* The usual site is near the apex of the left ventricle or in the septum. Degeneration, necrosis, and softening of the bloodless area follow. This softened area may bulge and form an acute aneurysm or may rupture. If small enough and under favorable circumstances it may be replaced by a fibrous mass.

THE MUSCLE-FIBERS.

(1) **Acute parenchymatous degeneration** or **myocarditis** may occur in acute pericarditis, endocarditis, or infectious fevers. It is the result of toxins. The *lesion* is usually general. The heart is soft, flabby, and pale on section, and the fibers contain granules that are not fat. The striations are less distinct or invisible. (2) **Fatty degeneration** is usually chronic, sometimes acute. It follows malnutrition in the aged, cachexias, wasting diseases, protracted fevers, anemias, phosphorus and some other poisons, pericarditis or hypertrophy and dilatation of the heart, and disease of the coronaries. The *lesion* is general or local, but usually in the left ventricle and papillary muscles. The degenerated muscle is soft, friable, grayish yellow or brown. The fibers contain granules or droplets of fat, being sometimes quite filled with them. (3) **Brown atrophy** is chronic. It is common in the aged. The muscle is brown, tough, and contains brown pigment granules around the nuclei. (4) **Hyaline transformation of Zenker**: In prolonged fevers, the fibers may be swollen and hyaline, the striations faint or invisible. (5) **Fragmentation and segmentation** are a separation of the fibers at the cement substance or a splitting transversely through the nucleus. It may be only a postmortem or preagonal change.

THE FIBROUS AND MUSCULAR TISSUE.

(1) **Acute Interstitial Myocarditis.**—This occurs in infections, especially diphtheria, typhoid, gonorrhea, and acute endocarditis and pericarditis. The *lesion* is acute granular, fatty, or hyaline degeneration of the muscle, with areas of small-celled infiltration of the connective tissue. It may lead to an abscess or to fibrous myocarditis. (2) **Fatty infiltration** (cor adiposus) is usually found with obesity. There is a complete thick layer of subpericardial fat. The fat extends inward among the muscle-fibers, may reach the endocardium, and be found in the papillary muscles. In extreme cases the muscle-fibers are atrophied and degenerated. (3) **Fibrous myocarditis** follows sclerosis of the coronaries; may extend from endocarditis or pericarditis; is more common in males and past middle life. It may be *partial*, in

the left ventricle near the apex, in the septum, or in the papillary muscles; or it may be *general.* New connective tissue occurs in grayish patches or streaks from microscopic size to ½ in. in diameter. There is hypertrophy of the other parts of the wall of the heart. The whole cavity dilates and the involved area may bulge as an aneurysm. *Microscopically,* there are the masses of new connective tissue and degeneration and atrophy of the muscle-fibers.

The **symptoms of myocardial disease** are notoriously inconstant and uncertain. There may be: (1) Cases of sudden death in persons in apparent health. The lesion is coronary sclerosis with or without infarct; or fibrous myocarditis; or fatty degeneration. (2) Sudden death in fevers. The lesion may be acute granular or fatty degeneration of the heart. (3) The *acute* or *subacute* degenerations may cause attacks of pallor, palpitation, syncope, and a rapid, weak irregular heart with signs of dilatation. The patients may recover, may die, or may go on to more chronic myocardial disease. (4) The more *chronic* cases of degeneration have symptoms of failure of compensation with signs of dilatation. Dropsy is rather rare. The pulse may be slow (40, more or less), somewhat irregular, there may be Cheyne-Stokes breathing, or pseudo-apoplexy. (This is an attack of vertigo and coma lasting from a few seconds to a few hours, the longer attacks may be followed by paralyses that last a day or two. Attacks are apt to recur every few hours for a few days. An attack may be fatal.) There may be angina pectoris, or less intense cardiac pain; or maniacal attacks. (5) Cases with arteriosclerosis and hypertrophy and dilatation have quite typical symptoms of loss of compensation.

On the whole, patients with myocardial change do badly. An accurate pathologic diagnosis is seldom possible.

Treatment.—The acute cases need rest in bed, and sometimes digitalis. In the more chronic cases treat the failing compensation. In all cases attack the cause if possible.

Aneurysm of the Heart.—(1) *Aneurysm of the wall. Acute* cases are due to necrosis or abscess. *Chronic* cases are due to stretching of a fibrous or fatty patch. Usually in the left ventricle near apex, there is a bulging of the wall. The *symptoms* are those of failing compensation or rupture of the heart, and occasionally the signs of a pulsating tumor projecting from the heart. Death is due to exhaustion of the heart or rupture. *Diagnosis* is very seldom possible. (2) *Aneurysm of a valve:* The valve, weakened by ulceration, bulges. Usually it is the aortic or mitral. This often ruptures, causing insufficiency with abrupt onset. The *diagnosis* is made at autopsy.

Rupture of the heart is due to fatty infiltration or degeneration, fibrous myocarditis, infarct, abscess or gumma. The rupture usually is induced by exertion. Its site is usually near the apex in the left ventricle. *Symptoms:* Sudden onset of cardiac pain, dyspnea, disturbed heart action, perhaps hemopericardium, and death in a few seconds or hours.

QUESTIONS.

Give the causes and lesions of coronary sclerosis.

Give the causes and lesions of acute parenchymatous degeneration of the heart.

Give the causes and lesions of fatty degeneration of the heart.

Give the causes and lesions of fibrous myocarditis.

Give the symptoms of disease of the wall of the heart.

What is the treatment?

Give the causes and symptoms of rupture of the heart.

NEUROSES OF THE HEART.

Palpitation.—*Etiology.*—(1) Hysteria, neurasthenia, gastric distention, anemia, or emotions. More common in females at puberty, the menopause, or perhaps menstruation. (2) A toxic form is due to tobacco, alcohol, tea or coffee. (3) Organic diseases of the heart.

Lesion.—There is none, but in protracted cases some hypertrophy of the heart may follow.

The *symptoms* occur only in paroxysms with abrupt onset. There are a consciousness of violent, rapid, and often irregular heart action; sometimes dyspnea, suffocation, sighing, and anxiety. On examination there may be pallor followed by flushing, the heart is often overacting, 120 or more; the sounds are loud, especially the second sound at the base, sometimes there is irregularity. In some cases with the same subjective symptoms the heart is slow and regular. The arteries pulsate unduly. There may be functional murmurs, or with heart lesions, organic murmurs. The paroxysm lasts a few minutes or hours and often is followed by the passage of much light urine. In some cases exertion aborts the paroxysm, in others it induces a return of symptoms. The paroxysms recur at varying intervals for a variable length of time.

The *prognosis* of an attack is very good, that of complete cure depends upon the causative condition.

Treatment is directed toward the cause. Sometimes strychnin, iron, or bromids, valerian, or hyoscyamus helps. The attack is treated by an ice-bag to the heart, rest or exercise, as the case may need. Perhaps morphin hypodermically may be needed in the worst attacks.

Paroxysmal Tachycardia.—*Etiology* is that of palpitation. There are paroxysms with an abrupt onset of very rapid heart action, 200 or more. In the typical cases subjective symptoms are slight or absent; in other cases they may resemble palpitation. The attack lasts a few minutes or an hour or two, and is apt to stop suddenly. The attacks return at intervals of days or weeks. Occasionally a patient dies in an attack. Treat like palpitation.

Bradycardia may be: (1) an idiosyncrasy in a perfectly healthy person. (2) It may occur in convalescence from acute fevers (pneumonia, typhoid, diphtheria, rheumatism). (3) In gastro-intestinal catarrh, ulcer or cancer. (4) Disease of the coronaries with fatty or fibroid heart, and in aortic stenosis. (5) Emphysema, asthma. (6) Anemia, gout, diabetes, myxedema. (7) Brain conditions, such as hemorrhage, meningitis, tumors, epilepsy, paresis, melancholia. The *symptom* is a slow heart action, 40 or less.

Stokes-Adams Syndrome.—In old men with advanced arteriosclerosis there are attacks of slow pulse (50 to 20), with syncope and sometimes epileptiform attacks.

ANGINA PECTORIS.

Etiology.—*Predisposing Factors.*—In males over forty; with hereditary tendency. There is coronary sclerosis and its sequels (myocarditis), or aortic insufficiency, or adherent pericardium in almost every case. The *exciting cause* is usually an exertion or emotion, sometimes gastric distention or exposure to cold.

The **symptoms** have an abrupt onset of pain in the heart, an excruciating sense of compression; the pain radiates to the left shoulder and down the arm, sometimes to the other shoulder and arm also. It is associated with an awful sense of impending death. There are often numbness of the precordium and hands, pallor, clammy sweat, and disturbed respiration. The heart action may be strikingly normal, but sometimes it is irregular and accelerated. The arterial tension is usually raised. The patient may be restless, or fixed and motionless. The attack lasts a few seconds or minutes and stops suddenly. After the attack the patient may feel shaky for a few hours or a day or two; he may belch up much gas or pass much pale urine. The paroxysm is apt to be repeated at longer or shorter intervals. Death in an attack is not rare.

The **prognosis** is not good. The heart lesion can not be cured, but the exciting causes may sometimes be controlled.

Treatment.—*In an attack:* Inhalation of amyl nitrite or chloroform; morphin hypodermically. *Between attacks:* Treat the primary condition, eliminate the exciting cause, administer arterial dilators in sufficient dosage to get the effect for long periods of time.

Pseudo-angina differs in the following points: It occurs at any age, especially in the neurotic and in women, and without discoverable exciting cause. There are no characteristic lesions. The attacks are often nocturnal and periodic. The pain is less intense, radiates less, and there is a sense of precordial distention. The patient is restless and emotional. The sense of impending death is absent. The attack may last from half an hour to several hours; it goes more slowly, and is not fatal. *Treatment:* In the attack morphin or inhalation of chloroform may be necessary. Treat also the nervous condition.

Vasomotor angina is a type of angina or pseudo- or toxic angina beginning with coldness and pallor of the extremities.

Toxic angina is severe cardiac pain due to tobacco or tea.

QUESTIONS.

Give the etiology, the symptoms, and treatment of palpitation.

Give the etiology and the symptoms of paroxysmal tachycardia.

Discuss the Stokes-Adams syndrome.

Give the etiology, the symptoms, the prognosis, and the treatment of angina pectoris.

Give the causes and the symptoms of pseudo-angina.

Give the differential diagnosis between angina and pseudo-angina.

DISEASES OF THE ARTERIES.

ARTERIOSCLEROSIS.

Etiology.—(1) A senile change that comes on earlier or later, depending upon the person's previous health, mode of life, and hereditary tendency. (2) In middle adult life or in the third decade arteriosclerosis may develop in those who have a strong heredity, or under the long-continued influence of alcohol, syphilis, gout, or lead; over-eating, excessive muscular exertion; chronic nephritis; or worry (all these conditions cause increase of blood-pressure). It is more common in males.

Lesions.—(1) **Nodular Form.**—The middle or outer coats of the artery show patches—usually around the vasa vasorum, perhaps beginning in their walls—in which there is swelling and hyaline degeneration of the muscle-fibers, often rupture of elastic tissue and then a small-celled infiltration, probably of new connective-tissue cells. This patch is weaker than the normal part of the artery. Opposite the patch, and by way of compensation, the intima becomes thickened from proliferation of the endothelium and connective-tissue. When the artery is distended its bore is smooth, but in the relaxed artery after death this area projects into the lumen as a gray "button." These areas in all of the coats tend to become yellow and soft from fatty degeneration and necrosis and contain, microscopically, crystals of cholesterin and fat acids, granules of fat, and detritus of disintegrated cells and fibers. Such a pasty mass is "*atheroma*." In the intima the necrosis may reach the surface and form an ulcer. The atheromatous mass may become calcified. Connective tissue forms around *it. Throm*bi often form on the ulcers, especially when lime salts are present. The fibrous areas may stretch and form *aneursyms.* There is hypertrophy and some dilatation of the left ventricle. This nodular form is usually found in the aorta on the longer curve of the arch, at the mouths of the branches (including the coronaries), and at the bifurcation. It is also common in the larger and middle-sized arteries, coronaries, cerebral, renal, temporals, radials, etc. It is rare in the visceral arteries of the abdomen. (2) **Senile form:** The arteries are dilated, lengthened, and tortuous. The atheroma, with ulceration, calcification, and thrombi is marked, so that the artery feels beaded or stiff. There is little hypertrophy of the heart. (3) **Diffuse form:** The artery is less elastic, is thick walled, and its lumen is lessened. *Microscopically,* there is hyaline or fatty degeneration or atrophy of the muscle-fibers of the middle coat. In the intima is new connective tissue which may show hyaline degeneration, so that the whole artery wall becomes homogeneous, thick, and hyaline, and the lumen small. The change begins in the capillaries and small arteries, attacks the whole circumference of the vessel, and is found everywhere in the body. There may be patches of atheroma in these arteries. Nodular arteriosclerosis of the larger arteries and aorta is also present, and often there is sclerosis of the aortic valves with insufficiency or stenosis. There is regularly considerable hypertrophy of the left ventricle and in time dilatation also. Fibrous myocarditis is rather common. (4) **Sclerosis of the pulmonary vessels** may follow protracted high tension in them (mitral lesions and emphysema). The arteries are most commonly involved, but the capillaries and even veins may be sclerotic. (5) Occasionally **veins** in which there is unusually high tension may be sclerosed, such as the tributaries of the portal vein in portal obstruction and the pulmonary veins mentioned above.

Symptoms.—The peripheral arteries look prominent and tortuous, and wriggle with the pulsation. They may feel beaded. The pulse fills slowly, falls slowly, is not large, and the rate may be slow. Considerable pressure is needed to obliterate the pulse and then the artery beyond can be easily felt. The tension is high, 130 to 200 mm. of mercury. *Heart symptoms:* There are the signs of hypertrophy of the left ventricle (see p. 250); the apex, 4 to 6 in. from the midline; a low rate; also very often a systolic murmur at the aortic area, transmitted upward. The accentuated aortic second sound is, of course, present. Later the signs of dilatation and the symptoms of failure of compensation appear. The results of coronary sclerosis (see p. 262) may be present.

Sclerosis of the cerebral arteries causes vertigo, Stokes-Adams syndrome, cerebral hemorrhage, embolism or thrombosis, transient attacks of hemiplegia, monoplegia, or aphasia. Coma may accompany these attacks or occur without them. General paralysis may follow.

Renal Arteries.—Sclerosis here results in interstitial nephritis.

Of the Limbs.—Thrombosis and gangrene, and the characters of the pulse.

Aneurysms are due to arteriosclerosis.

Course.—The patient may feel quite well for a long time, he may have some of the abrupt attacks from sclerosis of a group of arteries. Symptoms of several groups may coexist. In time symptoms of failure of compensation are apt to develop. The course may be modified by treatment begun early.

Treatment.—The causative factors must be eliminated if possible. The diet moderate and digestible. Life out of doors in an equable warm climate of low altitude is helpful. Drugs to control the tension are used—nitroglycerin, etc.—in sufficient dosage. Potassium iodid, 15 to 30 gr. a day for months or years, sometimes seems to help. The special symptoms must be treated.

QUESTIONS.

Give the etiology and lesions of arteriosclerosis.

Give the symptoms of sclerosis of the arteries of the heart, of the cerebral arteries, of the renal arteries.

How do you diagnose arteriosclerosis?

Give the general symptoms, course, and treatment of arteriosclerosis.

ANEURYSM OF THE ARCH OF THE AORTA.

Etiology.—Usually in men from thirty to fifty with the *co*existence of (1) a strain, either sudden or long-continued, and (2) arteriosclerosis in which syphilis is unusually common as a factor.

Lesions.—The most common form is sacculated aneurysm of the anterior wall of the ascending aorta and front part of the arch. Aneurysms of the transverse and descending parts of the arch are not rare; they are fusiform or sacculated. If the stretching involves the aortic ring there may be aortic insufficiency and a large heart; otherwise the heart is not much enlarged, but it may be displaced downward and to the left. The branches of the arch are often included in the aneurysm.

Symptoms.—**Pressure Symptoms.**—The *thorax* wall is atrophied and absorbed, with erosion of bone and cartilage. This causes severe constant boring pain at the upper sternum in front, or the fifth thoracic vertebra behind. *Superior vena cava:* In head, arms, and upper part of the thorax there are distention of veins, edema, cyanosis, clubbed fingers. An *innominate vein*, usually the right—symptoms as above, but only on *one* side. *Thoracic duct:* Loss of weight and strength; possibly chylous ascites. *Nerves.—Recurrent laryngeal:* Dry cough, hoarseness that may become extreme, and paralysis of the corresponding vocal cord (seen with the laryngoscope). The left nerve is pressed upon by aneurysms of the transverse arch, the right by those involving the right subclavian artery. *Brachial plexus:* Usually the right, pain in arm, and weakness of muscle. *Cervical sympathetic:* Midriasis and later myosis of pupil, possibly an early pallor and late flushing and sweating of the corresponding side of the head. *Intercostal nerves:* Severe protracted neuralgia, usually on the left side. *Spinal cord* (after erosion of the vertebræ): Possibly paralysis of the left leg or both legs. *Respiratory Organs.—Lungs:* The right apex in front, or the left lung below the apex behind, gives signs of collapse or compression. *Left bronchus:* The tracheal tug—a systolic pulling down of the larynx, felt by grasping the cricoid when the patient's head is moderately extended. Stridor, dyspnea, paroxysmal brassy cough, feeble breathing, and a tendency to bronchitis in the left lung are present. *Trachea:* Cough, stridor, and dyspnea as above. *Esophagus:* Rarely dysphagia is due to aneurysm.

Heart Symptoms.—There may be palpitation, dyspnea on exertion or orthopnea, or cardiac pain of any intensity.

Physical Signs.—(1) **Aneurysms Involving the Ascending and Transverse Parts of the Arch**—*Inspection:* Usually over the second and first right spaces, sometimes over the upper part of the sternum and even to the left is a pulsating area or a distinct pulsating tumor. *Palpation* may make out expansile pulsation over the tumor. There is a distinct diastolic shock, and sometimes a systolic thrill. *Percussion:* Marked dulness and increased resistance. The heart apex-beat may be normal or displaced downward and to the left. On *auscultation* at the above area there may be a systolic murmur, the second sound is usually very distinct and sharp. The heart-action is slow, often of good force, and regular. There may be also the signs of an associated aortic regurgitation. *The pulse:* With involvement of the innominate, and not of the left subclavian, the right radial pulse is much smaller and is later than the left; the carotids may show the same difference. With involvement of the left subclavian, and not the innominate, the left pulse is smaller and later. Often the femoral pulsation is indistinct. The tracheal tug may be present. (2) With **involvement of the descending part of the arch** there are in an area in the left interscapular region pulsation or a tumor with expansile pulsation; dulness, and a systolic bruit. The femoral pulse is small and delayed or absent.

The fluoroscope and *x*-ray photograph show the tumor. There are also physical signs of many of the effects of pressure.

Course.—The symptoms are often obscure or even absent. The aneurysms of the ascending part give the most evident physical signs, of the transverse part most pressure symptoms, of the descending part the signs are usually present, but are often misinterpreted. *Termination,* usually in rupture, externally, or into the trachea, bronchus, lung, or esophagus with escape of blood and death in a minute or two. For a few days there may be oozing of blood, forming a crust on the skin or leading to hemoptysis. It may rupture into the vena cava, pleura, pericardium, mediastinum, etc. Healed *aneurysmal sacs* have been discovered at autopsies, but the patients in whom the diagnosis is made clinically rarely live longer than two years.

Treatment.—Tuffnell's treatment is rest in bed and a very restricted dry diet for many weeks. Gelatin may be injected into the deep muscles of the gluteal region or elsewhere, 200 to 300 c.c. of 1 to 2 per cent. of gelatin in salt solution. Injections are repeated every few days for two or three weeks. Potassium iodid, 30 to 60 gr. a day for many weeks, electrolysis or the introduction of coils of wire may be tried. The symptoms may need treatment.

QUESTIONS.

Give the etiology, the lesion, and symptoms of aneurysm of the arch of the aorta.

Discuss the prognosis and treatment.

DISEASES OF THE BLOOD AND DUCTLESS GLANDS.

SECONDARY ANEMIA.

Etiology.—(1) Hemorrhage, acute or chronic, from any cause. (2) Starvation or inanition, from lack of food or inability to absorb it, as in obstruction of the esophagus or pylorus, or disease of stomach. (3) Excessive albuminous waste, as in chronic nephritis, suppuration, prolonged lactation, chronic diarrhea, etc. (4) From toxins in acute or chronic infections, or lead, arsenic, mercury, or phosphorus.

Symptoms.—After **acute hemorrhage** there is a rapid development of pallor, cold clammy skin, a soft pulse, becoming small and rapid, sighing breathing, darkness and swimming before the eyes, tinnitus, weakness, and perhaps syncope. If fatal, death may be preceded by convulsions. The *blood just after the bleeding* is pale and watery; the red cells are much reduced—1,000,000 to 2,000,000; the hemoglobin is correspondingly reduced; the color-index (percentage of hemoglobin divided by percentage of red cells) is 1; leukocytes somewhat increased; and the coagulation-time is much reduced. During *regeneration of blood* the red cells increase in number up to normal in from one to fourteen days, and normoblasts may be present; hemoglobin increases more slowly, the color index falls rapidly as the red cells increase and then rises slowly to 1. **Chronic hemorrhage** causes ordidary secondary anemia and regeneration is slower.

After **starvation** the *blood* is reduced in amount, the red cells may be normal or increased to 6,000,000 or even more.

The **ordinary symptoms of anemia** are pallor of skin and mucous membranes, but this is often deceptive; anorexia, indigestion, perhaps constipation; dyspnea on exertion which may be extreme; palpitation; the *heart* may be overacting or weak, it is accelerated, the first sound is shorter and higher pitched; *there* may be signs of some dilatation, and *hemic murmurs*—

viz., at the pulmonary area a systolic blowing murmur, often soft, but sometimes loud and rough; at the apex, a systolic blow; over the large arteries a systolic bruit; over the jugulars, especially on the right side, a systolic blow or, more typically, a continuous venous hum. The *pulse* is large and soft, sometimes a capillary pulse is seen. There are edema of the ankles at evening, or puffiness of the eyelids; a little fever at times; nervousness, listlessness, irritability, headache, fainting, neuralgia, tinnitus. The urine is pale and possibly albuminous. There may be amenorrhea, dysmenorrhea, leukorrhea, or less often menorrhagia. Strength is much lessened, but the anemia seems to cause no loss of weight.

The Blood.—*Mild Cases:* Red cells are a little smaller and paler, hemoglobin and color index somewhat lessened. *Moderate cases* show also some reduction in the red cells (4,000,000 per c.mm.), poikilocytosis, degenerations, and a slight leukocytosis. *Severe cases* show also greater reduction of red cells (2,000,000–3,000,000), normoblasts, an occasional megaloblast; the hemoglobin may be very low, 10 to 30 per cent., the color index quite low. During *regeneration* the red cells increase in number most rapidly, the hemoglobin more slowly, not reaching normal until long after all general symptoms have disappeared.

Prognosis depends upon the cause. The amount of blood for a fatal hemorrhage varies much with the individual and the general state of his health. It seems to be less in children and in men than in women.

PRIMARY ANEMIA.

A primary anemia is an anemia for which no adequate cause is discoverable.

CHLOROSIS.

Etiology.—In girls from fifteen to twenty-five years old. It is more common with a hereditary tendency from the mother, in blondes (?), housemaids, shopgirls working in bad air; with poor food; it may be induced by worry or a sudden intense emotion, or possibly by constipation. Some think it may be found in boys. There is a small group of cases with congenital hypoplasia of the arteries and usually of the genital organs also.

Symptoms are those of anemia (see p. 272). Special points: The skin may be greenish, but occasionally of good color with rosy cheeks; appetite is often perverted and capricious; gastroptosis and hyperchlorhydria are common, the stomach may be dilatated; attacks of intense gastric pain are not rare; constipation is the rule. The pulse usually is large and soft. Rarely there is thrombosis of a femoral vein or the longitudinal sinus (dulness, stupor, vomiting, midriasis, delirium, and perhaps double-choked disks). Hysterical symptoms are not rare. Uterine symptoms are very common. There is no loss of fat. The **blood** is pale and watery; red cells average 4,000,000, and seldom are below 2,000,000; they are pale, the average diameter is distinctly reduced; many microcytes are present; poikilocytosis and nucleated cells are rare. Hemoglobin averages 40 to 50 per cent.; is rarely below 25 per cent.; the color index is distinctly low, averaging 0.50; leukocytes are not much changed.

Course.—It usually develops slowly, often with lassitude and dyspnea on exertion; it lasts several weeks, the patient being bedridden only in severer cases. The patient feels quite well long before the hemoglobin reaches normal. Relapses are common. A sudden onset within a few days may occur, especially in a relapse and after a fright or other emotion.

Prognosis is good, except in the cases of aortic hypoplasia.

PERNICIOUS ANEMIA.

Etiology.—It occurs in middle life, less often in children, usually in males, with no discoverable cause. Hunter says it is due to absorption of toxins derived from pus from chronic suppuration about the gums (pyorrhea alveolaris), which has been swallowed. Very similar cases occasionally begin in pregnancy, or follow parturition or a severe hemorrhage, diarrhea, intense emotion, gastric atrophy, anchylostomiasis or bothriocephalus.

Lesions.—There is an excess of iron pigment in the liver, spleen, and kidney. There is hyperplasia of the bone-marrow, the yellow marrow becoming red. There may be fatty degeneration of the heart, liver, kidney, stomach, and intestine. Muscles are dark red, the fat is yellow and pale. There may be sclerosis of the posterior and lateral columns of the cord. Pyorrhea alveolaris is present.

Symptoms are those of a very severe anemia of fairly rapid course. Special points are the lemon-yellow skin; the pus around the roots of teeth; symptoms of gastric atony; occasional attacks of diarrhea; the heart symptoms are extreme dyspnea on slightest exertion; pulse 100 to 120, full and soft; a tendency to bleeding into the skin, mucous membranes, retina, etc. The edema may be marked and involve the serous sacs or lungs. Some fever is present at some time. There may be paresthesias, anesthesia, or spastic paralyses of the limbs. The urine is often dark from excess of urobilin, may contain a trace of albumin, and indican often is in excess. There is extreme weakness, the patient being bedridden after a time, but without emaciation. The **blood** is pale and limpid, and coagulates slowly. The red cells are usually 2,000,000 to 1,000,000 or even less; they look dark before and after staining; the average diameter is increased; there are many large cells, marked poikilocytosis, marked degeneration, *megaloblasts are present and often numerous*, normoblasts are less numerous. Hemoglobin is reduced, but the color index high, usually more than 1, and it may be 2 or more. Leukocytes are much diminished, chiefly the polynuclears, resulting in a relative increase of the other forms; eosinophiles may be slightly increased and a few myelocytes are common. During a remission the red cells increase to 4,000,000 or more; the color index may be less than 1, and there is often a leukocytosis.

Course.—The onset is rather slow, but sometimes the symptoms become very marked within a week; remissions with fair health for a few weeks or months are very common under treatment; relapses follow, however. The patients die of syncope, exhaustion, or cerebral hemorrhage. The average duration is about one year. Some cases seem to recover.

Treatment of Anemias.—**General Treatment.**—Liberal diet with red meat; fresh air; moderate exercise suited to the patient's strength; and laxatives when indicated. In **secondary anemia** treat the cause and give general tonics. Iron is of less definite benefit, but often helps. **Chlorosis**: Iron is specific. Blaud's pills, three a day; increased slowly to six or nine a day, continued until the hemoglobin is about normal, and then in decreasing doses for three months at least. The bad cases need two or three weeks in bed. **Pernicious anemia**: Arsenic, as Fowler's solution, 10 min. a day, or arsenous acid, $\frac{1}{15}$ gr. a day, increased to toleration, and continued indefinitely or for several weeks after apparent cure. It must be resumed with any evidence of a relapse. Rest in bed in every case, light nutritious diet, and fresh air are needed. Bone-marrow extracts and oxygen inhalations are recommended. Sometimes iron helps the arsenic.

QUESTIONS.

Give the etiology, symptoms, and treatment of moderate and of severe cases of secondary anemia.

Give the symptoms of a large, acute hemorrhage.

Give the etiology, the symptoms, prognosis, and treatment of chlorosis.

Give the etiology, lesions, symptoms, prognosis, and treatment of pernicious anemia.

Review the blood examination in secondary anemia, chlorosis, and pernicious anemia.

LEUKEMIA.

Etiology.—It is usual in middle life, but occurs in children; is more common in men. It may start in pregnancy or at the menopause, after malaria, or a blow over the spleen. The real cause is unknown.

Lesions.—**Splenomedullary Form.**—The spleen is very large—2 to 18 lb. and 6 to 12 in. long. There may be patches of perisplenitis or adhesions, the capsule often is thickened, the notches distinct, the vessels much dilated. In recent cases it is soft and may even rupture; in older cases, tough. The cut surface is brownish red, the malpighian bodies not visible; there are often grayish nodules of lymphoid tissue and sometimes hemorrhagic infarcts. Bone-marrow—the yellow marrow is greenish, or reddish brown, and may show *hemorrhagic* infarcts. The bone may be thinned and yielding. *Microscopically* there is hyperplasia of the lymphoid tissue and less fat, there are many nucleated red cells, mono- and polynuclear eosinophiles, and myelocytes, the cells often showing mitosis. The lymph-nodes show some enlargement from hyperplasia. **Lymphatic form**: The lymphoid-tissue everywhere is much increased from hyperplasia and shows mitoses in the lymphocytes. The lymph-nodes are large, soft, and do not coalesce; the follicles of tonsils, tongue, stomach, and intestines may be enlarged; sometimes the thymus and bone-marrow show the same changes. Similar tissue in abnormal sites may form *leukemic nodules* or tumors in liver or kidney, less often in the lung, testis, brain, etc. The liver is enormous (8 to 10 lb.), due chiefly to infiltration with lymphocytes in and around the capillaries, and leukemic tumors. The kidneys are large and pale, and show leukemic infiltration or tumors in the cortex. The spleen is usually moderately enlarged, with a similar lesion. In both types the heart and veins are distended with large, greenish or brownish clots, there may be Charcot-Leyden crystals in the blood. Emaciation is marked and there may be much dropsy. The blood-lesions are given below.

Symptoms.—There are all the symptoms of anemia, also vomiting and diarrhea, occasionally jaundice, usually attacks of fever of varying intensity (100°–104° F.), a marked tendency to bleeding in the skin, nose, alimentary canal, and sometimes into lungs, serous sacs, brain, and retina. Dropsy is common. Uric acid is in excess, priapism may occur, emaciation and weakness are marked. **Special Symptoms—Splenomedullary type**: The spleen is very large; its size may vary much within a few hours; often it forms a visible tumor, dull on percussion, with smooth surface; not tender; the notches usually are distinct; there may be a friction fremitus or a systolic souffle over it. It may displace the diaphragm and heart upward, or cause ascites. The liver is often moderately enlarged; the lymph-nodes may be enlarged. Rarely the sternum, ribs, and long bones of the limbs may be swollen and tender.

The Blood.—The drop may be quite opaque, and flows sluggishly. The red cells are moderately reduced in number—3,000,000, more or less; nucleated reds are abundant, normoblasts and also megaloblasts, often with polychromatic cytoplasm; hemoglobin is reduced; the color index usually less than 1. The white cells are several hundred thousands, 1 white to from 2 to 10 reds. The number fluctuates greatly within a few hours. From 20 to 50 per cent. are myelocytes, and this is the diagnostic point in the blood. Polynuclears and lymphocytes have a decreased percentage, but their actual numbers are much increased. Eosinophiles may be increased in percentage, the actual number is always much increased. During remissions the red cells may be about normal; the leukocytes are much reduced and may be nearly normal in number, but a differential count almost always shows the myelocyte excess and makes the diagnosis.

Lymphatic Form.—The superficial and deep nodes are much enlarged, soft, not adherent to surrounding structures, and do not coalesce. The liver is often very large; the edge is below the navel in the middle line. The spleen large, sometimes very large. *The Blood:* In the more chronic cases the red cells and hemoglobin are lower than in the other form. Nucleated reds, especially normoblasts, may be present. Leukocytes—50,000 to 200,000 are common counts; they average 1 white to perhaps 50 reds; lymphocytes, large and small, are very much increased, and constitute 90 to 98 per cent. of all the white cells. Myelocytes and eosinophiles may be present, but in very small percentages. Acute cases have severe anemia (perhaps 2,000,000 reds), and large lymphocytes are more numerous. **Mixed forms** of leukemia show combinations of the physical signs and blood characters of the two chief types.

Complications.—Tuberculosis, pneumonia, pleurisy, nephritis, dysentery, septicemia, and pyemia.

Course.—On the whole the splenomedullary form is more chronic, lasting, with remissions and exacerbations, for two or three years. The patients may feel quite well for a considerable time and come for treatment for a tumor in abdomen. Acute splenomedullary forms are very rare. Lymphatic cases are less common and, on the whole, less chronic, although some may last several years with the patient in apparent health. Acute cases may have noticeable symptoms for a *few weeks* or months only, and are more common in children.

The patients die of some complication or intercurrent disease, or of emaciation, diarrhea, hemorrhage, especially cerebral, of edema, or with high fever. Occasionally a case seems to recover.

Treatment.—Fresh air, nutritious food, mental and physical rest in bed for many weeks. Arsenic in large doses often helps. Quinin is given to the cases with malarial history, and iron to the anemic. Splenectomy is a serious operation, but some cases of recovery are reported after it.

QUESTIONS.

Give the etiology, the lesions, the symptoms, prognosis, and course of splenomedullary leukemia.

Give the same points of lymphatic leukemia.

Contrast the blood examination in the two forms.

Give the treatment.

PSEUDOLEUKEMIA (Hodgkin's Disease).

Etiology.—Usually in males in adolescence and adult life. Possibly it is predisposed to by syphilis, tuberculosis, malaria, or a large lymph-node from chronic infection from the nose, throat, or mouth.

Lesions.—The lymph-nodes enlarge and fuse into large nodular bunches which may have a firm capsule. At times they grow through the capsule and infiltrate the surrounding tissue. Rarely they suppurate or necrose. The histologic picture is like that of lymphatic leukemia. The lymphoid-nodules in the various organs show similar changes.

Symptoms.—The **blood** shows progressive secondary anemia, which may become extreme near death. The lymph-nodes form tumors that may give pressure symptoms. The cervical nodes usually are first attacked on one side, and then on the other. Later the axillary, inguinal, thoracic, retroperitoneal or mesenteric nodes become enlarged. They may form very large elastic masses, and are not tender. Symptoms of intrathoracic pressure are common (see p. 269, under Aneurysm), also dropsy of one or more limbs or serous sacs. Jaundice or bronzing of the skin may occur. The spleen is large, its edge several inches below the ribs, the liver may be moderately enlarged. Symptoms from lymphoid tumors in the central nervous symptom are rare, but there may be paraplegia, deafness, etc. Pain from pressure on nerves is common. *Fever* is present for weeks at a time. Emaciation and weakness are common. Bleedings may occur.

Course.—Usually lumps in the neck are first noticed, they gradually increase; other lymph-nodes enlarge and give their physical signs and other symptoms; anemia, with its symptoms, is more or less marked and progressive. Many cases show periods of remission of the symptoms and reduction in size of the tumors. Finally, after two or three years or longer, they die of exhaustion, asphyxia or other pressure symptoms, or of an intercurrent infection. Acute cases are rare, they die within two or three months, and fever usually is marked. Very rarely a case changes into one of lymphatic leukemia.

Treatment is like that of lymphatic leukemia.

QUESTION.

Give the etiology, lesions, symptoms, and treatment of Hodgkin's disease.

PURPURA.

This disease is a bleeding into the skin; the spots may be mere points (petechiæ), streaks (vibices), or larger spots or blotches (ecchymoses). They do not disappear on pressure; at first are red, then change color, through purple, brown, and yellows. One spot fades in from four or five days to two or three weeks, depending upon its size.

(A) **Secondary or symptomatic purpura** occurs in (1) infections such as septicemia, pyemia, malignant endocarditis, epidemic cerebrospinal meningitis and typhus; occasionally in other diseases. (2) In toxic conditions from snake poison, copaiba, quinin, belladonna, mercury, iodids, ergot, etc. (3) In malnutrition from anemia, tuberculosis, nephritis, cancer, pernicious anemia, leukemia, pseudoleukemia, *scurvy,* and very commonly in the aged. (4) In nervous conditions—locomotor ataxia, myelitis or hysteria. (5) From mechanical causes, such as a paroxysm of pertussis or convulsions.

(B) **Primary or Idiopathic Purpura.**—(1) **Simple purpura** occurs at puberty. There are crops of purpuric spots, usually on the legs, sometimes on the trunk or arms; often with diarrhea, but no fever. Occasionally there are joint pains also. Recovery follows in a week or ten days.

(2) **Arthritic Purpura.**—(*a*) *Peliosis* (*purpura*) *rheumatica* (Schoenlein's disease) is seen usually in males from twenty to thirty. Often it begins with sore throat and fever (101°–102° F.). In a day or two there is *arthritis* in several joints, with much pain and little swelling. The skin symptoms include purpura around the joints and on the legs, and often such conditions as urticaria, exudative erythema, edema in patches and, rarely, blebs with bloody contents. There may be albuminuria, endocarditis or pericarditis (rarely), or bleeding from mucous membranes. The patients usually recover in a week or two, but relapses and annual recurrences are common.

(*b*) *Henoch's purpura* occurs in children. *Symptoms:* The arthritis varies and may be very slight. The skin lesions include purpura and usually some form of erythema multiforme. There may be areas of edema. There are gastro-intestinal crises, with vomiting, diarrhea, and intense abdominal pain. These crises may precede the other symptoms by weeks. There may be bleeding from mucous membranes and kidneys. Relapses and recurrences are common. Most cases recover.

(C) **Purpura Hemorrhagica** (*Morbus maculosus* of Werlhof) is usually found in delicate girls. After a day or two of malaise the purpura appears and may be extensive, there may be large ecchymoses. Bleeding into the mucous membranes of the alimentary canal, into the lungs, kidneys, and central nervous system may occur. Anemia develops rapidly. There are prostration and fever of moderate degree. The mildest cases recover after one or two weeks. The severer cases, which are common, die in two or more weeks from exhaustion, anemia or cerebral hemorrhage. There are occasional fulminating cases that die within one to three days, often without bleeding from the mucous membranes.

Treatment of Purpura.—Measures to increase the coagulability of the blood—calcium chlorid, 40 to 80 gr. a day for three or four days; gelatin (2 per cent.) in salt solution, hypodermically, in doses of several ounces every two or three days (this is very painful). Other drugs used are suprarenal extract, ergot, etc. *Secondary cases:* Treat the primary condition. *Simple purpura:* Arsenic in large doses is given. *Arthritic purpura:* Salicylates are often given. Tonics are indicated in convalescence.

HEMOPHILIA.

Etiology.—It is strongly hereditary, being transmitted through the women (who are not themselves bleeders) to their male children, running thus through many generations. It is many times more common in males; usually appears in the first two years, less often as late as puberty. The affected families are often large, have a fine skin, and may be otherwise robust.

Lesions.—The smaller blood-vessels, especially the arteries, may have thin walls, due to deficiency of the muscular coat. There may be bleeding into and around the joints. The blood will be mentioned later.

Symptoms.—(1) *External bleeding* (the blood reaches a surface) from the nose, the alimentary canal, urethra, lungs, or less often the skin, eyelids, external ear, vulva, navel, etc. It is generally traumatic—after a scratch, blow, or slight operation (pulling a tooth, etc.). The bleeding is a capillary oozing, and persists for days or weeks. It may kill within a day. (2) *Internal bleeding* (the blood is confined) shows as petechiæ, ecchymoses, or, after a slight blow, as a hematoma. These are found in the skin and mucous membranes. (3) *Joint symptoms:* These may precede the bleeding. The joints, often the knee, are tender and swollen, from bleeding into them. This may come on acutely with fever. There may be later changes resulting in permanent deformity. (4) The *blood* coagulates slowly—ten to twenty minutes (the normal time is three to five minutes); the cells show the changes of anemia from hemorrhage (see p. 272).

Course.—If the bleeder does not die in the meantime, he is apt to lose the tendency to bleed at puberty or later. Many cases bleed to death from some trifling injury, but rarely from the first bleeding. Females are apt to have some menorrhagia for a few years, but they seem to stand pregnancy and parturition without undue loss of blood.

Treatment.—*Preventive:* Avoid carefully all wounds and operations. *During the bleeding—Locally:* Pressure, turpentine, tannic acid, suprarenal extract or gelatin (5 per cent. solution). For nose-bleed—inhalation of carbon dioxide; swabbing with fibrin ferment and calcium chlorid; plugging the nostril. *Internally:* Calcium chlorid; gelatin, hypodermically or by mouth or rectum; tincture ferric chlorid, ½ dr., every two hours, with sodium phosphate; ergot, liquid extract of hydrastis, 1 dr. daily, and many other drugs are used. During convalescence treat the anemia.

SCURVY (Scorbutus).

Etiology.—Theories: (1) It is a specific infectious disease due to an unidentified organism. (2) It is due to the lack of salts from the food; either of the base—potassium; or of the acids—lactic, malic, citric. (3) It is an intoxication from decomposed food, especially meat.

It formerly was very common among soldiers and sailors. It is endemic in certain districts in Europe; may occur in epidemics in a community living in some improper way. Sporadic cases occur. Predisposing factors are bad hygiene, cold damp quarters, exposure, lack of fresh vegetable food, mental distress or depression, or excessive exercise. Starvation alone does not cause it. It attacks all ages.

Lesions.—Fatty and granular degeneration of the liver, heart, and kidneys; hemorrhages into the skin, mucous membranes, solid viscera, joints, serous sacs, etc.; a large soft spleen; the lesions of the gums and the anemia.

Symptoms.—There are emaciation, weakness, anemia, but no leukocytosis. The skin is dry and gray. The *gums* are sore, swollen, spongy, and bleed easily; the teeth are loosened; the breath foul; the tongue swollen; the appetite poor; constipation. There is *bleeding* into the skin and mucous membrane, with petechiæ, ecchymoses, or hematomata; into the kidneys and elsewhere. Nervous symptoms are those of great depression; later there may be delirium. The temperature is normal or subnormal, unless there is some inflammatory process. The urine is dark, heavy, often albuminous, and sometimes bloody. Joints are acutely tender and may be swollen. The epiphyses may separate, bones may necrose, and subperiosteal bleeding may cause tumors.

Course.—The patient grows steadily weaker, and without treatment dies of exhaustion, pneumonia, dysentery, or bleeding into the pleura, lung, or cranium. With treatment recovery usually follows in all but the most advanced cases.

Treatment.—*Of the Attack.*—Two ounces of lemon-juice or vinegar daily, and a diet of fresh meat, vegetables, and fruit cure promptly. The mouth should be treated with dilute carbolic or other antiseptic mouth-wash; the constipation, by enemas. *Prevention* is most important. When it is impossible to get fresh or canned vegetables and fruits, 1 oz. of lemon-juice or vinegar should be taken daily.

INFANTILE SCURVY.

Etiology.—It occurs in children, from the seventh to the fourteenth month, that have been fed for several months on sterilized milk, condensed milk, or proprietary foods. It is very rare in breast-fed babies. The causative factors in the food are not known.

Lesions.—There is bleeding under the periosteum of the bones of the legs and thighs, also around the joints and into the muscles, skin, mucous membranes, kidneys, etc. The bones are swollen from subperiosteal bleeding, usually at the lower end of the femur and near the ankle. There may be diastasis of the lower ends of the femur and tibia. The joints are usually normal. Other lesions are mentioned below.

Symptoms.—The gums near the teeth are soft, swollen, tender, and bleed easily. There may be hematomata over teeth that are not yet through. Diarrhea is common. The *legs are* very painful on motion or handling. In marked cases the limb is never moved voluntarily, and paralysis may be suspected. There is swelling over the lower end of the femur and tibia, sometimes extending along the whole shaft. The limb may be edematous. Diastasis may be found late in the disease. There are symptoms of the bleedings mentioned above, with increasing anemia, emaciation, and fretfulness, and often albuminuria.

Course.—A gradual onset of pain in the legs and fretfulness, and in about a week the gum symptoms. This, with some emaciation, makes up the milder cases. Later, there may be the bleedings into the periosteum and elsewhere, the intense leg symptoms, marked emaciation and anemia. Finally, if untreated, after two to four months, the baby may die of malnutrition, pneumonia, colitis, or possibly of bleeding. Rickets is often associated with scurvy.

The **prognosis** is good with proper treatment in all but the most advanced or the complicated cases.

Treatment is very simple: A suitable diet of modified fresh cows' milk alone usually cures. The cure is much facilitated by orange-juice, ½–4 oz., daily. Meat juice is given. The anemia and other symptoms should receive appropriate treatment.

QUESTIONS.

Give the varieties of purpura, and the etiology of symptomatic purpura.

Give the symptoms of simple purpura, of peliosis rheumatica, of Henoch's purpura, of purpura hemorrhagica.

Give the treatment of purpura.

Give the etiology, lesions, symptoms, course, and prognosis of hemophilia.

Give the etiology, pathologic theories, lesions, symptoms, prognosis, and treatment of scurvy.

Give the etiology, lesions, symptoms, and treatment of infantile scurvy.

STATUS LYMPHATICUS (Constitutio Lymphatica, Lymphatism).

Etiology.—It is found in children and babies in unfavorable surroundings. The parents may give a history of the same condition. The efficient cause is quite unknown.

Lesions.—There is hyperplasia of lymphoid tissue where-ever found. There is enlargement of the lymphoid organs and of the thyroid. There may be hypoplasia of the heart and aorta. Rickets is often present.

The **symptoms** are those of the large tonsils, adenoids, and lymph-nodes. The thymus gives dulness over the upper sternal region. The most striking symptom is sudden death in anesthesia, even after the first few whiffs of chloroform, or less often, under other conditions, such as the prick of an injecting needle or other trifling operation, immersion in cold water, or possibly after acute fevers.

The tendency is usually outgrown during the second decade.

QUESTION.

Give the etiology, lesions, and symptoms of the status lymphaticus.

ADDISON'S DISEASE.

Etiology.—This is most common in males, from twenty to forty; it may follow a blow on the abdomen or back, or caries of the spine or tuberculosis elsewhere.

Lesions.—The suprarenal gland on one or both sides is usually tuberculous and shows caseation, tubercle tissue, and connective tissue. Occasionally it is atrophied with or without interstitial inflammation. Very rarely it shows malignant disease or hemorrhage. The ganglia of the solar plexus usually show degeneration or atrophy of the nervous tissue, and sclerosis. This may occur without adrenal lesion. Other lesions are mentioned with the symptoms.

Symptoms.—(1) **Pigmentation.**—The skin is dusky, bronzed or nearly black. This change is early, and always most marked on the exposed parts of the body, the places normally pigmented, and at points of pressure. It may be quite general. There may be darker mole-like spots, or occasionally white patches of leukoderma. The mucous membranes of the mouth and elsewhere are pigmented. (2) **Asthenia** of the heart and other muscles, and of the brain, causes dyspnea and palpitation on slight exertion; a rapid weak heart; small soft pulse; often functional murmurs; *dizziness,* *syncope,* tinnitus, and headache. The patient is easily and constantly tired. Lack of mental vigor, slow speech, and, later, somnolence are present. (3) **Gastrointestinal symptoms:** Anorexia and attacks of vomiting, lasting a few days, are early and constant. Attacks of abdominal pain (crises), and, later, of diarrhea are common. Emaciation is not marked; there is more or less anemia, but it is rarely extreme. There may be a positive tuberculin reaction.

Course.—The onset is insidious; the weakness, heart and stomach symptoms, come on gradually; later the pigmentation appears. There may be periods of marked remission for weeks or months. Usually, within a year the patient dies of asthenia or syncope. Some cases live for five years or more or seem to recover. There are (rarely) acute cases with vomiting, diarrhea, delirium, convulsions, coma, and fever lasting a few months or weeks, with little or no pigmentation.

Treatment.—Suprarenal gland by mouth may be tried. Besides this, use general hygienic, dietetic, and tonic treatment, with much rest in bed.

QUESTION.

Give the etiology, lesions, symptoms, prognosis, and treatment of Addison's disease.

SPLEEN.

Temporary enlargement of the spleen occurs in most infectious diseases and in toxemia to a varying extent. The spleen is **permanently enlarged** (although its size may fluctuate), in chronic malaria, leukemia, pseudoleukemia, cirrhosis of the liver, splenic anemia, syphilis, and amyloid disease. *Physical signs:* On *inspection* there may be a fulness or distinct tumor in the left side of the abdomen that may move with respiration. On *percussion*, with the patient on the right side, the splenic dulness may extend above the seventh intercostal space and in front of a line from the left sternoclavicular joint to the tip of the tenth rib. On palpation in deep inspiration the edge of the spleen may be felt beneath the left rib cartilages. Larger spleens may give a smooth tumor, extending from beneath the ribs, which may go beyond the middle line or reach the pelvis. It is usually movable. Its edge is sharp or rounded, hard or soft, and the notches can be felt.

Wandering or **floating spleen** may be due to a congenitally long peritoneal fold, or the structures at the hilum may be stretched by the weight of a large spleen. It may be a part of general splanchnoptosis. The movable spleen is usually large. The edge may be just palpable under the ribs; or the whole spleen may be below the ribs and freely movable, and there is tympany over the usual site of splenic dulness. Other symptoms may be absent or there may be a feeling of weight, pain or aching in the splenic region. Rarely the pedicle may become twisted. This causes acute, intense pain, much swelling and tenderness of the spleen, with high fever and sometimes collapse. This may soon be followed by necrosis of the spleen, general peritonitis, and death. *Treatment:* A movable spleen may be held in place by pads and a binder. It may be sutured in place, with deliberate injury to the peritoneum, to favor adhesions. Exceptionally it may be excised. For a twisted pedicle immediate laparotomy and excision are necessary.

Rupture of the spleen is a rare accident in malaria, typhoid, splenic leukemia, or abscess or infarct of the spleen, usually following a blow over it. The *symptoms* are sudden pain over the spleen and the symptoms of internal hemorrhage, with blood in the peritoneal cavity and often death. The *treatment* is immediate laparotomy and excision of the spleen.

Infarct of the spleen may be due to (*a*) *embolism* in typhoid, endocarditis, aortitis, pyemia or disease of the splenic artery or (*b*) to a *thrombus* in the splenic vessels, especially in septicemia and pyemia. The infarct may be simple or infectious. It is conical, usually white, but sometimes engorged with blood (hemorrhagic infarct). Ultimately, if the patient lives, a scar forms. There is localized peritonitis at the surface of the infarct. If infected, an acscess forms. *Symptoms* are often unnoticeable. In other cases there is sudden pain at the site of the infarct with tenderness, a peritoneal friction sound and usually slight enlargement of the spleen. **Abscess of the spleen** is usually due to an infectious infarct; it may *lead to* septic peritonitis.

Splenic anemia is a condition that has not yet been thoroughly worked out. Its cause is unknown. It usually occurs in adult males. There is a very large spleen, which may reach the middle line. Following this, is an anemia of secondary type, often with leukopenia, a tendency to hemorrhages, and a very slow course lasting years. Toward the end there may be added a large cirrhotic liver and its symptoms (this syndrome is called Banti's disease). There may also be ascites without cirrhosis. The patient dies of an intercurrent disease or asthenia. The treatment is the usual treatment for anemia and excision of the spleen.

QUESTIONS.

Give the causes and physical signs of enlarged spleen.

Give the causes, symptoms and physical signs, and treatment of wandering spleen, of rupture of the spleen, of infarct and abscess of the spleen.

Give the symptoms of splenic anemia.

What is Banti's disease?

DISEASES OF THE THYROID GLAND.

GOITER.

Etiology.—Goiter is rare in this country, it is endemic and common in certain countries, especially in Switzerland. It is more common in women and soon after puberty. It may be hereditary. It is supposed to be due to (1) an infection or (2) certain waters from magnesium limestone which, however, are made harmless by boiling.

The **lesion** results in enlargement of the thyroid, which may be slight or marked, and general, or limited to any part of the gland. Several varieties are described. (1) **Simple or parenchymatous**: A hypertrophy and hyperplasia of all the tissues of the gland. (2) **Cystic**: Dilatation of the alveoli, which may run together and form large cavities. In these two forms the alveoli contain colloid or mucinous liquid which is sometimes bloody or brown. (3) **Vascular or pulsating**: Due to dilatation of the blood-vessels. (4) **Fibrous**, with marked increase in the connective tissue. **Mixed forms** are very common.

Symptoms.—There is a tumor on one or both sides, or in the middle of the neck, corresponding to the thyroid. Its size varies extremely. The largest goiters overhang the sternum. It moves with the larynx and trachea in swallowing, is not adherent to other structures, is often lobulated, and may fluctuate in places. Its growth is usually slow, but is often accelerated at menstruation or pregnancy. The superficial veins may be distended. The goiter may press upon the trachea, the recurrent laryngeal nerve, esophagus or veins. There may be a systolic bruit over it. The general health is not much affected. Rarely there may be sudden death.

Treatment.—In goiterous districts, drinking-water should be boiled. Thyroid extract by mouth, or thyroidectomy may be tried. Iodin externally and by mouth is recommended. Some cases recover spontaneously, especially when they move from the affected district.

QUESTION.

What are the causes, the varieties, the physical signs, and treatment of goiter?

EXOPHTHALMIC GOITER (Parry's, Graves', or Basedow's Disease).

Etiology.—It is much more common in females; in the third decade; with a neuropathic heredity; or with a heredity of this disease. It may follow anemia, debility, severe protracted emotional states, especially with depression. It is possibly due to over-activity of the thyroid with an increased or perhaps perverted secretion.

Lesions.—The thyroid is enlarged. There is an increase in the size and number of the cells of the alveoli. The blood-vessels are dilated. The thymus persists and is enlarged from hyperplasia.

Symptoms.—There are four cardinal groups of symptoms—(1) **Circulatory**: There are palpitation and breathlessness on exertion or emotion, a rapid pulse, perhaps 90 to 100 at rest, and much higher under excitement; throbbing *arteries* and a capillary pulse; a forcible apex-beat; loud heart sounds, often a systolic murmur at the base, the apex and over the large arteries. Later there may be symptoms and signs of dilation and hypertrophy of the heart with irregularity. (2) **The thyroid symptoms**: There is a goiter that is seldom very large; it may pulsate visibly, and over it there is a systolic or continuous thrill and bruit. Its size may fluctuate rather quickly. It is not tender. (3) **The eye symptoms**: Exophthalmos of varying extent and usually bilateral. Von Graefe's sign—as the eye looks downward following a moving object the upper lid follows sluggishly or incompletely. Stellwag's sign is due to spasm of the upper lid causing widening of the palpebral fissure—a rim of white sclerotic shows above and below the iris; also winking is infrequent. In marked cases the cornea may ulcerate. The eyes tire easily in looking at near objects. (4) **The nervous symptoms**: A fine involuntary tremor that is constant, but increases with excitement. Pronounced neurasthenic or hysteric symptoms are present.

The other symptoms that may be present are flushing of the skin, sweating, especially of the hands and feet, some pigmentation of the eyelids, face, neck and other places, transient patches of edema, a capricious appetite, marked thirst. There may be attacks of vomiting or of diarrhea, or of both, lasting for a week or two. Fever is sometimes present. The urine is usually normal. Emaciation and loss of strength vary with the acuteness of the other symptoms, but often they are marked.

Course.—Acute cases are rare. They may last from a few days to a few months, and sometimes are fatal. The **chronic cases**: The cardiac and nervous symptoms usually appear first, the exophthalmos and goiter appear later. The disease runs along with longer or shorter exacerbations and remissions. It lasts several years and may end in recovery or in more or less improvement, which is apt to be interrupted by a relapse. Rarely it gradually changes to myxedema. Other cases die in acute gastro-intestinal attacks, or of syncope, emaciation or asthenia, or of some intercurrent disease. Perhaps half the cases recover, either completely or with some permanent exophthalmos or goiter.

Treatment.—The diet should be simple and digestible, without alcohol, tobacco, tea or coffee. The patient should lead a quiet life with exercise proportionate to her strength. During an exacerbation the patient should stay in bed for many weeks. Thyroidectomy or bilateral excision of the cervical sympathetic may cure. Other remedies are cold to the neck, digitalis, strophanthus, belladonna, iron, arsenic, and glycerophosphates. Galvanism may be used with the anode at the back of the neck and the cathode over the cervical sympathetic.

QUESTIONS.

Give the etiology and the lesions of exophthalmic goiter.
What are the four most important groups of symptoms?
Give the physical signs and the other symptoms.
Give the prognosis and treatment.

MYXEDEMA.

Etiology.—Mostly in women of middle age. Perhaps there is a hereditary tendency in women to this or to exophthalmic goiter. It is rarely the termination of exophthalmic goiter. Probably it is due to absence of thyroid secretion.

Lesions.—The thyroid shows atrophy of the epithelium, sometimes with evidence of inflammation or tumor. The areolar connective tissue everywhere in the body is thickened and contains much gelatinous mucin-like substance (myxedema). The fat may seem increased.

Symptoms.—The entire body looks swollen, the skin is dry, rough, feels doughy, but does not pit on pressure; the hair is thinned, is dry and brittle; the hands broad and "spade-like"; the expression stupid, the features heavy, the tongue thick and swollen; the voice hard and halting; the temperature subnormal; there is a tendency to bleeding from the mucous membranes. Nervous symptoms are marked—slowness of intellect and of sensation, depression, irritability; the patient is suspicious of everything; there may be melancholia. The gait is sluggish and somewhat atactic. The urine is diminished, contains less nitrogen, and sometimes is albuminous. The weight may increase, but the strength is lessened.

Course.—The onset is slow, often with a gain of weight. The symptoms grow more and more pronounced, and may last five to fifteen years. The patient may die of exhaustion or insane, or often of an intercurrent disease, especially pneumonia or tuberculosis.

The **prognosis** is usually good in the early cases properly treated. Within a few weeks the patient loses the superfluous weight and becomes quite normal. She will remain so if treatment is continued indefinitely. The later or very severe cases improve more slowly and perhaps incompletely.

Treatment.—Thyroid gland (dried, raw, slightly cooked, or extracts of it) is given at first in small doses (half of a sheep's thyroid or 5 gr. of dried gland or extract, three times a day), increased to the point of toleration ; continued a little below this for weeks or months until the patient is well, and then in smaller dose throughout life. Excessive dosage is shown by "thyroidism" (sweating, flushing, vomiting, rapid heart, palpitation, some fever, headache, vertigo, nervousness, perhaps syncope).

Cretinism is endemic in goiterous regions. Sporadic cases occur rarely anywhere. *Lesions:* The thyroid is absent, or the epithelial cells are atrophied, although the gland may be large from fibrous tissue. The patient is a myxedematous dwarf. *Symptoms:* Sometimes the symptoms are congenital, but they usually appear about the sixth to twelfth month. Some cases develop a few years later. The child is stunted ; has a coarse, thick face ; looks fat but pasty ; has a large belly and short legs. The tongue is swollen and may protrude constantly, the teeth appear late and decay early, speech is slow and defective, the voice harsh and lifeless ; there are all grades of mental deficiency, often complete idiocy. The child develops slowly for fifteen years or so and then seems to remain in the same state indefinitely. *Prognosis:* Untreated it is hopeless. If treated very early the patient may become quite normal in every way. If treatment is stopped the symptoms return. If treatment is begun late the outlook is doubtful, although some improvement is usual. *Treatment:* Thyroid gland.

Operative myxedema (cachexia strumipriva) is a myxedema that develops in persons whose thyroid is atrophied from disease or wholly removed by operation. If a piece of functionating thyroid or accessory thyroids are present the condition does not develop.

QUESTIONS.

What is the etiology of myxedema, its lesions, symptoms, prognosis, and treatment?

What are the symptoms of thyroidism?

Give the etiology, symptoms, prognosis, and treatment of cretinism.

What is operative myxedema?

DISEASES OF THE KIDNEY.

MOVABLE KIDNEY.

Etiology.—This is more common in women and on the right side, often as a part of splanchnoptosis. It may be due to the disappearance of perirenal fat, increased weight of kidney, tight lacing, or congenitally lax peritoneal attachment, with long renal artery and veins.

Symptoms.—Very often no subjective symptoms are present, but there may be aching and dragging in the loin, neuralgia of the lower intercostals, neurasthenic and dyspeptic symptoms, and a tendency to mental depression. *Physical examination:* Possibly, there may be in the loin a slight depression, and a tympanitic note when compared with the other side. Upon deep inspiration the lower end of the kidney may be felt, this is called *palpable kidney.* In other cases, on deep breathing the upper end of the kidney may be caught by the fingers; the kidney slips up as soon as the fingers release it (*movable kidney*). Or the kidney may be felt and moved around freely, reaching sometimes the middle line or the iliac fossa (*floating kidney*). The kidney is not very tender, but pressure may cause a sickening feeling. Less common symptoms—**Deitl's crises**: Attacks of chills, fever, nausea, vomiting and collapse, with severe abdominal colic. The urine is diminished and contains an excess of urates and sometimes of oxalates, possibly some blood or pus. Dilatation of the stomach, attacks of jaundice, edema of lower limbs or intermittent hydronephrosis occasionally occur.

Treatment.—Increase the fat. Hold the kidney in place by a properly adjusted abdominal pad and belt. Nephrorrhaphy or even nephrectomy may be needed.

QUESTION.

Give the causes, symptoms, and treatment of movable kidney.

HEMATURIA.

Causes.—(1) **General and blood diseases**: Hemorrhagic forms of the infectious diseases, intense or protracted jaundice, purpura, scurvy, hemophilia, malaria or leukemia. (2) **In the kidney**: (*a*) Acute congestion due to acute inflammations, either Bright's or suppurative; or to poisons such as carbolic acid, turpentine, cantharides; rarely to a floating kidney. (*b*) Infarcts. (*c*) Tuberculosis. (*d*) Neoplasms. (*e*) Parasites: Bilharzia, echinococcus, filaria. (*f*) Trauma, from a blow, a kidney stone or operation. (*g*) Apparently idiopathic. (3) From the **ureter, bladder, prostate** or **urethra,** with trauma from instruments, calculi or blows; inflammations, parasites, tumors; or from varicose veins of the bladder.

Symptoms.—Blood in the urine is determined by the presence of clots or red cells, hemoglobin, and albumin. **Source of bleeding**: (*a*) If the *kidney* the blood and urine are well mixed. If bleeding is very profuse there may be blood-clot casts of the pelvis or of the ureter. Blood-casts of the kidney tubules may be present. The urine is red or brownish, there may be a brown sediment. (*b*) From the *ureters*—casts of the ureter are common. (*c*) From the *bladder*—the blood may appear with only the last portion of the urine. Bleeding may be profuse and, after standing, large clots may form. (*d*) *Urethra*—the blood appears with the first portion of the urine or independent of micturition. Cystoscopy may determine the exact source of the blood in the lower passages or the ureter through which the blood reaches the bladder.

Treatment.—Rest in bed, dry cupping or cold to the loins, ergot, opium, gallic or tannic acid, or hydrastis, by mouth. The primary condition may be amenable to treatment.

HEMOGLOBINURIA.

This results from hemoglobinemia, which is the existence of hemoglobin in solution in the plasma. (1) **Toxic hemoglobinuria**: From poisoning by carbolic acid, potassium chlorate, phosphorus, arseniuretted hydrogen, carbon monoxid, pyrogallic acid; poisonous or tainted fungi; the hemorrhagic forms of infectious diseases (malaria and yellow fever are the *most important*); perhaps scurvy or purpura. It may follow extensive burns or the infusion of blood from another species. There is an epidemic hemoglobinuria of the newborn. In general, exposure to cold or wet or undue exertion may favor it. (2) **Paroxysmal hemoglobinuria** is a rare condition, usually found in adult males, apt to be induced by exposure to cold or wet and by exertion. It seems at times to be associated with Raynaud's disease.

Symptoms.—The urine is dark brown, red or almost black and contains albumin. The pigment is usually methemoglobin, it may be hemoglobin, or both may be present. These are identified by the spectroscope, the guaiac test, or the potassium hydrate test. The urinary sediment is granular and dark, and may contain an insignificant number of red cells. In paroxysmal hemoglobinuria there are attacks of hemoglobinuria lasting a day or two, sometimes with chills, fever, nausea and vomiting, diarrhea, lumbar pain, possibly jaundice.

Treatment.—External warmth, hot drinks, perhaps amyl nitrite.

ALBUMINURIA.

Etiology.—(1) **Kidney lesions**: Bright's disease; acute or chronic congestion; amyloid, granular or fatty degeneration; suppuration or tumors of the kidney. (2) **Without obvious kidney lesion**: (*a*) Blood conditions- chronic lead, mercury or arsenic poisoning; severe anemia, leukemia, scurvy, purpura, syphilis; with excess of urobilin, of glucose or bile pigment; in pregnancy, diabetes, or after etherization. (*b*) After an epileptic fit, head injury, apoplexy, or in tetanus. (*c*) Febrile albuminuria in any fever, but especially the infectious diseases. (*d*) Cyclic albuminuria, usually in adolescent males, is a transient albuminuria recurring daily for a few hours; it may follow meals, exertion or emotion. Most cases are slight and recover; some develop nephritis. A few casts are sometimes found. (*e*) Orthostatic albuminuria—albumin appears after the patient stands for a few hours. (*f*) Albumin may appear after an undue exertion, emotion, or after an excessive albuminous meal. Groups (*d*), (*e*), and (*f*) are sometimes called physiologic or functional albuminuria. (*g*) Albumin may be derived from pus or blood from any part of the urinary tract (spurious albuminuria). The urine contains serum albumin associated with smaller amounts of serum globulin.

INDICANURIA.

Indican is indoxyl potassium sulphate; it is derived from the decomposition of albumin, and is present in the urine in health, being least with a milk diet, and most with a meat diet. It is increased with increased decomposition (1) in the small intestine, especially in intestinal obstruction, acute or chronic peritonitis, or acute or chronic gastritis with decrease of hydrochloric acid; (2) with increased decomposition elsewhere, as in large collections of pus (empyema), or gangrene, especially of the lung. Indican is not in excess when only the colon is involved.

PYURIA.

Pus is derived from suppurative inflammation of any part of the urinary tract, from an abscess rupturing into it, or from leukorrhea. The urine contains albumin, pus cells, and usually many bacteria. If alkaline and ammoniacal there is cystitis; if acid, cystitis may be present or absent. Cystoscopy may help find the source of the pus.

Chyluria.—(1) Due to filaria or (2) non-parasitic. The urine is milky from the presence of fat globules. It may clot. It may contain blood also.

Choluria.—Bile constituents are present (see Jaundice, p. 177).

Glycosuria.—(1) **Persistent** glycosuria is found with diabetes mellitus, including pancreatic disease, and certain brain lesions, especially in the floor of the fourth ventricle. (2) **Transitory**: (*a*) Drugs—phlorizin, morphin, alcohol, chloral, chloroform, amyl nitrite, sulphuric acid, mercury, carbon monoxid, etc. (*b*) Nervous causes—paresis, epilepsy, dementia; emotions, especially worry; after fractures of the skull, concussion or hemorrhage of the brain; disease or injury to the bulb; possibly after any general convulsion. (*c*) Alimentary tract—gastric catarrh, functional disorder of *the liver,* excessive ingestion or imperfect digestion of carbohydrate food. (*d*) Infectious diseases. (*e*) In gout or obesity.

Lithuria.—Uric acid is probably derived from the nucleins of the tissues and food. It is **decreased** in chlorosis, many chronic diseases, and during and before an attack of gout. It is **increased** in pernicious anemia, leukemia, fevers, diabetes, in the "uric acid diathesis," during digestion, especially of nitrogenous food, after exercise. The normal amount is 1 gm. or less daily (about $\frac{1}{50}$ of the amount of urea excreted). It exists in the form of urates, almost never as free acid. On standing it may be precipitated as urates or free uric acid. The precipitation depends upon several factors, and is no measure of the amount of uric acid present. Occasionally it is precipitated within the body and passed as gravel, or it may form a stone. The symptoms of the "uric acid diathesis" are those of irregular gout.

Oxaluria.—Calcium oxalate may precipitate from normal urine on standing, but the number of crystals is not a good guide to the amount excreted. It is in excess after certain foods (apples, pears, rhubarb, asparagus, tomatoes, etc.). Also in "idiopathic oxaluria," a condition marked by indigestion, neurasthenic symptoms, depression or even hypochondriasis and persistent oxaluria. It may form calculi.

Phosphates.—Normally about 2 to 5 gm. of phosphoric acid are excreted daily, but the phosphatic sediment is not a measure of the amount excreted. It is increased in wasting diseases, such as phthisis, leukemia, severe anemias, acute yellow atrophy. In phosphatic diabetes it may amount to 7 to 9 gm., and is associated with thirst, polyuria, and wasting. Phosphates (even when in normal amount) may be precipitated in alkaline urine and form a stone, usually in the bladder, and associated with cystitis.

Leucin and **tyrosin** are not found in normal urine; they result from albuminous decomposition, and are found in acute yellow atrophy, phosphorus poisoning, less often in typhoid fever, small-pox, yellow fever. They are present in certain cases of vomiting of pregnancy, and seem to be important.

Hematoporphyrin is found in poisoning from sulfonal, trional, tetronal, and some other conditions. The urine is reddish and gives a characteristic spectrum.

Urea.—Normally about 30 gm. (450 gr.) are excreted daily. It is increased after proteid food, exercise, in diabetes and fevers. It is lessened in nephritis, liver diseases, cachexias, anemias, inanition, etc.

Acetone may occur in traces in healthy urine, also in fevers, cachexias, starvation, and after anesthesia, but chiefly in diabetes. It gives the urine a fruity ethereal odor.

Diacetic acid is pathologic; it may occur in fevers, but chiefly in diabetes.

Beta-oxybutyric acid may occur with diacetic acid. It is perhaps the cause of diabetic coma.

QUESTIONS.

Give the causes of hematuria.

How may the source of the blood be inferred?

What is the treatment?

What are the causes of hemoglobinuria; the diagnosis and the differential diagnosis from hematuria?

What are the causes of albuminuria?

What causes an excess of indican in the urine?

What causes pyuria?

Give the causes of glycosuria.

What causes increased excretion of urates, oxalate of calcium, phosphates, and urea; a decreased excretion of urates, phosphates, and urea?

What is idiopathic oxaluria?

When are leucin and tyrosin found in the urine?

What is the significance of acetone, diacetic acid and β-oxybutyric acid in the urine?

ANURIA (Suppression of Urine).

Etiology.—The secretion of urine may cease temporarily or permanently. It occurs (1) **in intense renal congestion** from any cause: (*a*) In fevers, especially cholera, yellow fever; (*b*) in nephritis; (*c*) after cantharides, phosphorus, lead, turpentine. (2) There may be **complete mechanical obstruction,** as when both ureters are plugged by stones, or the ureter of a solitary kidney is obstructed by stone or twisting or kinking, or a solitary kidney is removed by operation. There may be obstruction in the urethra or bladder. (3) **Reflex anuria** may follow operations or injury to one kidney, especially its removal; or to any part of the urinary tract, even catheterization; or, very rarely, an operation anywhere, or anesthesia; or it may follow the plugging of one ureter only by a stone. (4) Anuria may occur in **hysteria.**

Even in fatal anuria there are usually no symptoms for a time. Then there are weakness, collapse and death after four to fourteen days. Less often there may be uremic symptoms.

Prognosis.—If the cause can not be removed the patient dies. Suppression for a few hours or a day or two is common and may be followed by no bad results.

Treatment.—Surgery may attack the cause of obstruction. To start and increase the secretion of urine the loins may be cupped or treated with hot fomentations; other measures are the hot pack, hot-air bath, pilocarpin, purging with calomel, salines or other drugs, hot, protracted irrigations of colon, diuretics, saline infusion, milk and water diet.

QUESTION.

Give the etiology, symptoms, prognosis, and treatment of anuria.

UREMIA.

Etiology.—This term connotes a great variety of symptoms that occur in Bright's disease or, exceptionally, with complete or partial suppression of the urine. Its real cause is not known, it is variously supposed to be due to: (*a*) Retained excrementitious matter that the kidney should eliminate; (*b*) to perverted metabolism, resulting in abnormal compounds that act as poisons; and (*c*) to edema of the brain.

The **symptoms** that are included are the following: The skin is usually pale, dry and hard; occasionally there may be sweating, with a white frosty deposit of urea; a tendency to eczema. Dropsy may be present or absent. Uremic stomatitis: the gums and mucous membrane of the mouth are red and swollen; there may be salivation; the tongue is swollen and heavily coated, there is difficulty in chewing and swallowing; the breath is foul and of a somewhat urinous smell. There may be indigestion, hiccough, persistent vomiting, regardless of eating. The vomitus may be watery and of urinous odor. There may be diarrhea, with small serous stools, perhaps of urinous odor. The heart is slow and forcible, and the pulse of high tension. Later, the heart often becomes weak and irregular, and the pulse small and soft. The blood contains an excess of urea. Temperature varies; it is often subnormal or normal; after convulsions or with inflammations there may be fever; often there is antemortem hyperpyrexia. Dyspnea is common, either constant or paroxysmal, especially nocturnal and with orthopnea (uremic asthma); Cheyne-Stokes breathing is common in coma and may occur without coma. Nervous symptoms are very important—headache, often occipital; neuralgia, vertigo, often rather persistent; paresthesias, pruritus, cramps in limbs; active delirium, restlessness, sleeplessness, occasionally acute mania or delusional insanity; or dulness, apathy, low, muttering delirium, stupor, coma. There may be twitching of muscles, or partial or general epileptiform convulsions which may be repeated frequently (eclampsia). Following the convulsions and coma, or occurring without them, there may be uremic amaurosis, deafness, aphasia, monoplegias, or hemiplegia. These paralyses may last a few hours or days, but usually pass off. The urine varies, but it usually is diminished and contains more albumin and casts than formerly. Besides this there is a tendency to certain inflammations, often as the terminal infection, such as pneumonia, pleurisy, pericarditis, peritonitis, meningitis, croupous colitis.

Any case may show a few or many of these symptoms, and it may run an acute or chronic course, so, clinically, the cases vary extremely.

Acute Uremia.—A very important group is that with coma. It may develop in a person giving active symptoms of Bright's disease, occasionally with suppression, but very often in chronic interstitial nephritis while the patient seems quite well. Some of the milder symptoms, such as vertigo, headache, gastro-intestinal disturbance, or high-tension pulse may precede. Suddenly the patient falls unconscious, often with epileptiform convulsions. The face may be pale or somewhat flushed, the heart slow and forcible, the pulse hard, breathing stertorous, often Cheyne-Stokes or otherwise irregular, the *breath may be* urinous. The temperature after convulsions is often elevated (103°–104° F.), or it may be normal. The pupils may be dilated or contracted, coma is usually very deep, and reflexes are abolished; if the coma is less deep, reflexes are usually exaggerated. Urine is retained, diminished, and contains more albumin and casts than before; there may be suppression. The convulsions may be repeated; the coma may last only a few minutes or several days. Recovery is not rare, it may be quite prompt and complete or there may be as sequels paralyses of sight, hearing, speech, or limbs that may pass off in a short time. In other cases the coma persists or there may be periods of stupor, the heart weakens, the pulse becoming small, soft and fast. The patient dies, sometimes in a few hours, or not for a week or ten days. The immediate cause of death may be heart failure, cerebral hemorrhage, pneumonia, or other terminal infection, or edema of the lungs. Dropsy may be slight or absent. The cases must be diagnosed from other comas.

Chronic uremia is very common in chronic exudative nephritis. There may be, first, the gastro-intestinal symptoms or high-tension pulse, or uremic asthma and dyspnea, a temperature low or often subnormal, or the lesser nervous symptoms. Dropsy usually is marked. To the first symptoms various others are added, and, finally, the patient becomes dull, apathetic, stupid, comatose, and dies of exhaustion or a terminal infection.

Prognosis.—Many cases recover from an attack of acute uremia, but the prognosis of the original disease remains. Chronic cases are usually fatal in a few weeks or months if the symptoms are pronounced. The insanities sometimes recover.

Treatment.—*Coma with high tension:* Venesection (1 to 2 pints) may be followed by prompt return of consciousness. Elimination should be aided by purging with croton oil, 2 gtt., or calomel, 5–10 gr.; a hot pack, repeated twice a day, or hot-air bath, and by other measures to increase the secretion of urine. *Convulsions* may be controlled by chloroform. The diet should consist of milk and much water. *Symptomatic treatment* and the treatment of the primary cause are indicated. Morphin is safe and efficient for restlessness and headache if the tension of the pulse is high.

QUESTIONS.

Give the etiology and the symptoms of uremia in general.

Give the symptoms of an acute attack of uremia with coma and convulsions.

Discuss chronic uremia.

Give the treatment of uremia.

CONGESTION OF THE KIDNEY.

Acute Congestion (Active Hyperemia).—**Causes:** From cantharides, turpentine, cubebs, copaiba; chilling of the skin; operations or anesthesia; at the onset of acute nephritis; possibly in fevers. **Lesions:** The kidney is large, red, its capsule not adherent, its cut surface is very red and drips blood. The capillaries in the tufts and elsewhere, and the veins are distended with blood, the epithelium may show granular degeneration. **Symptoms:** The urine is diminished, of dark color, high specific gravity (1020 or more), contains a trace of albumin, some hyaline casts and red cells. It may be suppressed for a few hours. There may be some lumbar pain, malaise, and slight fever. It lasts a day or two and, unless nephritis follows, the kidney and urine become normal. **Treatmont:** Rest in bed and the treatment for anuria (see p. 301).

Chronic Congestion (Passive Hyperemia).—**Etiology:** That of venous stasis, which may be (1) *general* in chronic heart-inadequacy from any cause, or chronic lung disease, such as emphysema, interstitial pneumonia, and chronic adhesive pleurisy. Or (2) *local,* from cirrhosis of the liver, tumors in abdomen (pregnant uterus, etc.) or ascites pressing on the vena cava or renal veins; rarely from thrombosis of renal veins. **Lesions:** The kidneys are large and dark red, the stellate veins are distinct; the capsule is not adherent, except in long-standing cases, when it adheres in patches. The kidney cuts with resistance, is tough, firmer, its cut surface dark red or purplish, the markings are coarse, the veins are engorged. *Microscopically,* the capillaries of the tufts and elsewhere are distended with blood, their walls often thickened. The epithelium is often degenerated and swollen. Later some tufts are more or less replaced by fibrous tissue and there are other lesions of interstitial nephritis. **Symptoms:** The urine is diminished, dark in color, of high specific gravity, deposits urates on standing, contains a trace of albumin, some hyaline casts, and at times a few red cells. The urea is not decreased. There are usually no other kidney symptoms, but as interstitial nephritis develops so does the chance of its symptoms. *Treatment* is that of the primary condition.

QUESTIONS.

Give the causes, symptoms, and details of the treatment of acute congestion of the kidneys.

Give the causes, lesions, and urinary symptoms of passive hyperemia of the kidney.

RENAL DROPSY.

The dropsy of renal disease may have an abrupt or slow onset; it may be slight or involve all parts of the body; it may be well marked in certain places and slight elsewhere. In general it appears first in the face, especially the eyelids, it then involves the feet (often the dorsum of the foot), the penis and scrotum, or labia, the dependent parts of the trunk, the upper and lower extremities, the serous cavities, brain and all parts of the body. The most dangerous sites are the throat (edema of the glottis), the brain, the lung and the serous sacs. When slight it is apt to appear in the recumbent position, and to lessen or disappear when the patient gets up and moves about. When pronounced it is constant, but tends to be more marked in the dependent portions of the trunk or limbs. The edematous parts are pale, swollen, sometimes extremely so, the skin may even crack and ooze serum; in places like the penis, scrotum or throat the tissues may be translucent. There is pitting on pressure. The liquid in the serous sacs is usually light in color, of a specific gravity less than 1015, and contains but little albumin and few cells.

QUESTIONS.

Give the symptoms of renal dropsy.

Give the differential diagnosis between an exudate and renal dropsy of the serous cases.

ACUTE NEPHRITIS.

(Acute Bright's Disease, Acute Exudative Nephritis, Acute Diffuse Nephritis, Acute Parenchymatous Nephritis.)

Etiology.—It is more common in children and the first half of adult life, and in males. Alcoholism, acute and chronic, seems to predispose. It may follow exposure to cold and wet, especially when drunk; extensive burns and certain skin diseases, especially the erythema group. It complicates acute infections, such as scarlatina, diphtheria, yellow fever, cholera, and rarely other infections. It may be a primary infection. It may follow cantharides, turpentine, carbolic acid, iodoform, mineral acids, potassium chlorate, phosphorus, lead, mercury, arsenic, or ether inhalation. It occurs in pregnancy, especially the later months. It may follow injury to the kidney.

Lesions.—The kidney may be swollen: in early stages it is acutely congested (see p. 304). The capsule is not adherent; on section the cortex is swollen, pale (or congested, and the glomeruli may stand out prominently), the finer markings are blurred, the medulla is red. *Microscopically.—The glomeruli:* The capillaries show swelling and proliferation of the endothelium, contain blood-cells, endothelium, and thrombi. They may show hyaline degeneration. The epithelium covering the tufts and that of the capsule is swollen, proliferated and desquamating. The cavity of the capsule contains epithelium, white and red blood-cells, and albuminous clots. The connective tissue of the tuft shows exudative inflammation and, at times, productive inflammation with proliferation of the connective-tissue cells, and later formation of fibrous connective tissue. The *tubular epithelium* is swollen, opaque, its striations and nuclei become indistinct, it may desquamate. The tubules contain casts. The *stroma* is the seat of exudative inflammation, the leukocytes tending to collect in foci. It may show productive inflammation also, with areas of proliferation of connective-tissue cells and later of fibrous tissue. Other lesions are mentioned with the symptoms.

Symptoms.—(**A**) The more characteristic cases, such as those following exposure, may begin suddenly, perhaps with chills or chilliness, occasionally with convulsions, followed by a few days of fever (101°-103° F.). The skin is pale and dry, sweating is slight. *Dropsy* (see p. 305) may be marked within twenty-four hours; prostration varies, there may occasionally be aching or pain in the loins; the heart is slow and forcible, the aortic second sound accentuated and the tension high; the urine is decreased (10 to 16 oz. a day), or suppressed for a few hours or a day, it is dark, often smoky, with much sediment, and is acid, 1020 or more, contains much albumin (¼ to 1 per cent. by weight), urea is decreased in total output. *Microscopically* there are many hyaline, granular, epithelial, and blood casts; red and some white blood-cells, and renal epithelium.

Course.—(1) After the abrupt onset the symptoms are pronounced for a few days, anemia develops rapidly, then the urine increases, the albumin decreases in percentage, the dropsy clears and the patient may feel quite well, even before the dropsy has disappeared. The albumin and casts gradually lessen, but are present for two to four weeks after the other symptoms have gone. Finally, recovery may be perfect and the kidney left undamaged. (2) The onset may be less abrupt, and some cases may be more protracted, becoming subacute or chronic (these are the cases with productive inflammation). (3) At any time dangerous symptoms may arise, acute dilatation of the heart, edema of the glottis, lungs, brain, or large transudation into the serous sacs, acute uremia or secondary infections, usually pneumonia, or actual inflammations (not merely dropsy) of the serous sacs or meninges. However, most of this group also recover.

(**B**) After scarlet fever or diphtheria, cases develop in convalescence or late in the disease that are very like the above; they are, however, apt to be rather protracted, and to have the slower onset.

(**C**) Sometimes in these diseases and usually in the other infections the nephritis is more insidious, there may be no dropsy, but the patients become paler and sicker, the urinary changes are present, and death may follow acute uremia or a terminal infection.

(**D**) In pregnancy the onset is slow, dropsy is common, and eclampsia is the chief danger.

Treatment.—Rest in bed; protection from chill by woolen blankets; milk and much water, purging with salines daily; measures to produce sweating, the hot pack, hot-air bath, perhaps pilocarpin; cupping or heat to the loins. When the patient improves, the diet should be increased slowly, and meat is the last thing to be added. He should be carefully protected from exposure to cold. Saline diuretics may help, or digitalis if the blood-pressure is low, or diuretin. Iron helps the anemia. The dropsy may need paracentesis or incisions of the skin. Uremia needs its suitable treatment, as do the other symptoms and complications and the primary disease. Removal to a warm equable climate helps the protracted cases.

QUESTIONS.

Give the etiology and lesions of acute nephritis.

Give the symptoms, course, and prognosis of the ordinary cases.

Give the symptoms of the less typical cases occurring with certain infectious diseases.

Give the treatment of acute nephritis.

CHRONIC PARENCHYMATOUS NEPHRITIS.

(Chronic Diffuse Nephritis with Exudation, Chronic Exudative Nephritis, Chronic Bright's Disease.)

Etiology.—It may follow acute nephritis, especially in scarlet fever and pregnancy. Often it begins insidiously, perhaps some of these cases are secondary to the infectious fevers, especially malaria. Tuberculosis, syphilis or chronic suppuration may precede it. Beer, spirits, and working in cold, damp places seem to predispose. It is more common in adults before middle life, but is not rare in children. More males are affected.

Lesions.—The **large white kidney** is large, pale and yellowish, smooth, the stellate veins may be prominent. The capsule is sometimes adherent in places. On section the cortex is thickened and yellowish-white, but there may be opaque areas, the medulla is congested. *Microscopically:* The *glomeruli* show proliferation and swelling of the epithelium of the capsule, of that covering the tuft, and of the *endothelium*. Some show all degrees of atrophy ending in a fibrous nodule. The *convoluted tubules* vary in different kidneys and in different parts of the same kidney. Some are atrophied ; others have flattened epithelium and a dilated lumen, which may contain casts ; others have swollen, opaque granular or especially fatty epithelium. There may be desquamation or hyaline degeneration. The *arteries* are thickened. The *stroma* shows patches of round-celled infiltration and connective tissue. The **small white kidney** (pale granular kidney) is probably a later stage of the large white kidney in most cases. The kidney is small, granular, pale or mottled, the capsule thickened and somewhat adherent. The tissue is firmer and cuts with some resistance. The cortex is thinned, with yellowish-white areas, sometimes with red areas also. *Microscopically*, the changes are like those of the large white kidney, but there is relatively more connective tissue which often has contracted, more atrophy of the other structures, and more arteritis. **Chronic hemorrhagic kidney** is similar to the large white kidney, but is mottled with red or brown areas that represent bleeding in the stroma around the tubules and glomeruli. In all there is more or less general arteriosclerosis and hypertrophy and dilatation of the heart.

Symptoms.—(1) Some follow acute nephritis. The symptoms abate, but the anemia, some dropsy, and albumin and casts persist, and the heart symptoms become marked. (2) Probably most cases have an insidious onset. There are at first rather persistent gastro-intestinal disturbances, headache, and some failure of general health and strength. After weeks or months of this condition the more characteristic symptoms develop. These are chiefly anemia and pallor, dropsy, the urinary and the cardiovascular changes. The skin is pale and waxy, the anemia progresses slowly, and may be extreme. There are the symptoms of hypertrophy of the left ventricle with accentuated second aortic sound, the pulse is of high tension. The dropsy is constant and persistent. At first it is slight (see p. 305), but usually becomes very marked. The urine is more or less diminished, of dark color with heavy sediment, 1020 or higher, contains much albumin, 1 to 3 per cent. (¼–¾ by volume), the urea is decreased. *Microscopically* there are casts—hyaline, granular, fatty ; fatty epithelium, leukocytes, and urates. The loss of strength continues and the patients become bedridden. The disease drags on with exacerbations that are quite like acute nephritis, and remissions in which the anemia, albumin, casts and dropsy persist. Sometimes even these clear up for a time. The patient is liable to uremia (see p. 301). Chronic and acute uremia are common, but attacks of sudden coma and of convulsions are rather rare. There may be albuminuric retinitis. After six months to two years the patient may die of exhaustion, dropsy, chronic uremia, some terminal infection (inflammation of the serous sacs or meninges or pneumonia) or of failure of compensation of the heart. A few cases, usually children, after a year or less gradually get well and seem to remain so. Some cases run along with the exacerbations and remissions for a long time, three to five years or even more, and their symptoms grow more like those of chronic interstitial nephritis, the dropsy disappearing or becoming very slight, the urine increasing to more than the normal 50 ounces a day, becoming paler, of lower specific gravity (1010), the albumin and casts nearly or entirely disappearing for long intervals, the hypertrophy of the heart and arteriosclerosis becoming more marked. These cases die like cases of interstitial nephritis, or in a tardy exacerbation of exudative type.

Treatment.—In the *exacerbations:* As an acute nephritis. In the *remissions:* The patient should live out of doors in a warm, equable climate. Diet should be moderate, with little meat and much water. The uremia, dropsy, high tension, etc., need their appropriate treatment. Diuretics are often used with some benefit.

QUESTION.

Give the etiology, lesions, symptoms, course, prognosis, and treatment of chronic parenchymatous nephritis.

CHRONIC INTERSTITIAL NEPHRITIS.

(Chronic Bright's Disease, Chronic Diffuse Nephritis without Exudation, Contracted Kidney, Gouty Kidney, Sclerotic Kidney, Arteriosclerotic Kidney.)

Etiology.—It shares the etiology of the other chronic *productive* inflammations (see p. 266). Primary cases develop before middle life, even in adolescence. Secondary cases are usually in middle life and after. In both groups it is more common in males, with a hereditary tendency, syphilis, perhaps malaria, with gout, chronic lead poisoning, lithemia, overwork (both muscular and mental), worry, overeating, especially of meat, steady drinking. It follows chronic congestion of the kidney, hydronephrosis or chronic pyelitis.

Lesions.—The kidneys usually are small, may weigh an ounce or two apiece, hard, nodular, may show cysts containing a watery or clotted liquid, are reddish or gray or mottled. The capsule is adherent. On section the kidney is tough. The cortex is thin (in places $\frac{1}{12}$ inch), the pyramids also are somewhat smaller. *Microscopically:* The *glomeruli* vary—some have disappeared, some have thick capsules or thick capillaries, often hyaline; the connective tissue is increased, some being represented by little fibrous nodules. After chronic congestion the capillaries usually persist and are dilated. The *tubules:* In the masses of connective tissue they may be obliterated, or smaller and contain casts, the epithelium is flattened. Between the masses of connective tissue they may have flattened or swollen, granular or fatty, epithelium. They may be dilated, some forming cysts. The *stroma* shows connective-tissue proliferation, this at first is quite cellular, later fibrous. It occurs in patches in the cortex and also diffusely among the tubules of the cortex and in the pyramids. The *arteries* are prominent and thickened with arteriosclerosis, they may show hyaline changes. Besides the kidney changes there are general arteriosclerosis, hypertrophy and dilatation of the heart, first of the left ventricle, but later general and often extreme, often cirrhosis of the liver and emphysema.

Symptoms.—**Urine.**—Increased (80–120 oz. a day), pale, 1010, often less; albumin a trace, but it may be absent for days or weeks, urea is usually decreased, a few hyaline casts are present. Acute uremia in all its many phases is common. The ingravescent chronic uremia ending in death is rare. **Eye:** Besides uremic amaurosis without lesion, there may be dimness or disturbance of vision from albuminuric retinitis, with flame-shaped hemorrhages, or yellowish patches around the papilla or macula lutea; or distended or tortuous veins. Pallor and anemia are less marked than in parenchymatous nephritis, loss of flesh and strength are common. There are thirst and polyuria. Dropsy of renal origin is not common, but occasionally edema of the glottis or lungs comes on. There may be exacerbations that resemble exudative nephritis.

The symptoms of the associated lesions are most important —the hypertrophy and dilatation of the heart, the arteriosclerosis, the emphysema, with its chronic bronchitis, the cirrhosis. Then, too, there may be an intercurrent disease, especially pneumonia.

Course.—The lesion may last for a long time (years) without causing any noticeable symptoms. There may be a series of attacks of uremic symptoms, mild or severe, or of other symptoms lasting a variable time. Between attacks the patient may feel well or he may have general symptoms of failing health, which sometimes become worse after each attack. He usually has symptoms from the associated lesions. In spite of all this he may live an active life for five to fifteen years. Death is often due to acute uremia, sometimes the first attack of uremia ; or it may be due to edema of the glottis or lung, to pneumonia or other intercurrent disease, or to the associated lesions.

Treatment.—The general treatment as in the other chronic nephritis, and treatment for the uremic and other symptoms.

QUESTIONS.

Give the etiology, lesions, symptoms, prognosis, and treatment of chronic interstitial nephritis.

Review the urinary symptoms of acute nephritis, chronic parenchymatous nephritis, and chronic interstitial nephritis.

AMYLOID KIDNEY.

Etiology.—That of amyloid disease in general (see p. 197). It may be associated with chronic nephritis, especially of the parenchymatous variety.

Lesions.—The kidney is large or perhaps of normal size, the stellate veins may be prominent. The capsule may not be adherent, the cut surface shows the cortex to be thickened, pale, and waxy, and it gives the amyloid reactions. The *pyramids* are red. *Microscopically*, there is thickening and amyloid change, first in the capillaries of the tufts, then the efferent vessels, the straight vessels, the capillaries and veins around the convoluted tubules, the basement membrane of the tubules and possibly of the epithelium of the tubules. The vessels of the pyramid may be involved to a less extent. There are often the changes of the large white kidney. There is amyloid in the liver, spleen and perhaps intestines.

Symptoms.—Besides those of the causative condition, and amyloid disease elsewhere, renal dropsy is often present and may be marked. The urine varies, but in the more typical cases it is increased (80 oz.), pale, 1010 or less, contains considerable albumin with an unusual percentage of globulin; many hyaline and granular casts. Waxy casts are of no diagnostic importance. At times or in some cases the amount may be normal, the albumin little or even absent for considerable periods, and the urine may be further modified by the parenchymatous nephritis that may be present.

The **prognosis** is bad. The kidney does not recover.

Treatment.—That of the causative condition and of chronic nephritis.

QUESTION.

Give the etiology, lesions, and urinary symptoms of amyloid kidney.

NEPHROLITHIASIS.

Etiology.—Usually before fifteen or after fifty years, more in males, and with a sedentary life. The stone is formed by the aggregation in the pelvis of the ureter of substances precipitated from the urine. The precipitation is due partly in some cases to an excess of the substance, but chiefly to conditions of the urine making it less soluble. The aggregation seems to take place around a nucleus of bacteria, mucus, coagulated albumin or blood.

Varieties.—(1) **Uric Acid.**—Usually mixed with urates, sometimes in strata (urates may occur alone in children). This forms (*a*) urinary sand, a coarse, hard powder; (*b*) gravel, larger grains; (*c*) stones of considerable size, ¼–½ in. in diameter, which may be single or multiple, are smooth, round or somewhat irregular, hard, dense, and often stratified ; or (*d*) dendritic stones, like the above, but large branching masses, that may form perfect casts of the whole or part of the pelvis and calyces. (2) **Calcium oxalate,** or **mulberry stones,** often contain uric acid also, and are rough, warty, very hard and dense and very dark. (3) **Phosphatic stones** are composed of calcium or ammoniomagnesium phosphates, often mixed with calcium carbonate, and they may have a uric acid stone as a nucleus. They are white, friable, are formed in alkaline urine, and are more common in the bladder. (4) Other stones are described, but they are uncommon.

Other Lesions.—Pyelitis is more or less marked; there may be suppurative pyelitis, pyelonephrosis, or hydronephrosis. In time there is sclerosis of the kidney.

Symptoms.—(1) **Renal Colic.**—The classical case is due to the passage of a fair-sized stone from the pelvis of the ureter to the bladder. The onset is abrupt, sometimes following a muscular strain. There is pain in the loin of the affected side, intense, agonizing, with some tenderness, the pain radiates along the ureter, and to the testis, which is retracted (or to the labium), along the inner side of the thigh, often to the flanks or sometimes to the lower part of the thorax. The patient is pale, covered with cold sweat, may vomit, has a rapid weak heart and small pulse, a chill, some fever (103° F.), more or less collapse, and may faint. There may be frequent painful micturition with bloody urine. The pain lasts a few hours, or with remissions or even intermissions for a few days, and stops suddenly. The other symptoms then quickly subside. The attack may be followed by the passage of bloody urine for a few days or by the passage of large amounts of clear pale urine. The kidney may be tender and sore, the testis swollen and painful.

Other Cases and Symptoms.—*Gravel* and *sand* may be passed for long periods of time with no other symptoms. A *stone in the pelvis* may cause no symptoms, or there may be aching or pain in the loin, or bloody urine at intervals and lasting a variable time, or attacks like renal colic. There may be pyelitis or the symptoms of the other renal changes. There may be periods of intermittent fever with chills, like *malaria,* but sometimes with hematuria or pyuria. A *stone* *impacted in the ureter* may stop the urine from its kidney and sometimes reflexly from the other kidney also, with even fatal anuria. It may cause hydronephrosis, or repeated attacks of more or less modified renal colic. A stone when it reaches the bladder runs the course of vesical stone. Attacks of renal colic may be repeated at intervals for years or there may be but a single attack. The general health often remains quite good. Occasionally an attack of renal colic is fatal. The associated inflammations may be fatal in time or cause marked ill health. *X*-rays help in the diagnosis of certain stones.

Treatment.—**Renal Colic.**—Morphin hypodermically to control the pain. Sometimes chloroform inhalation. Hot baths, much hot water by mouth, hot protracted colon irrigation. **Between the attacks** try to lessen the precipitation by giving much water; also with uric acid stones by alkalies, by diet and mode of life to lessen uric acid formation. Possibly piperazin or urotropin may help. With phosphatic stones remove the alkalinity of the urine by benzoic or boric acid or saccharin. Calcium carbonate may help both types. Operation may be indicated.

QUESTIONS.

Give the etiology, the varieties, and general characteristics of urinary calculi.

Give the symptoms of renal colic.

Give the other symptoms of nephrolithiasis.

What is the treatment?

HYDRONEPHROSIS.

The **cause** is urinary stasis, unilateral or, rarely, bilateral. (1) The ureter may be plugged by a stone, by fragments of cancer, cheesy masses (tuberculosis, inspissated pus), blood-clots, pus from the pelvis or kidney, or a mass of parasites. It may be stenosed by a scar from an ulcer, or congenitally, or by twisting or kinking in movable kidney, by pressure from tumors, ascites, peritoneal exudate, or adhesions. (2) In the bladder there may be tumors, scars, paralysis, enlarged prostate. (3) There may be stricture of the urethra. Hydronephrosis is more common in adult males.

Lesions.—The pelvis, the ureter above the obstruction, and the kidney also are dilated. In a well-marked case the kidney is reduced to a thin shell of connective tissue. The whole tumor forms a smooth or lobulated cyst which may weigh several pounds. The contained liquid is yellowish, often clear, of low specific gravity, often alkaline; may contain a little albumin, and often some urea or uric acid and urinary salts. It sometimes is turbid with epithelium and pus cells, or bloody or colored by pigment from blood (red or brown). Occasionally it is thick and gelatinous.

Symptoms.—(1) **Congenital Cases.**—A large tumor may obstruct labor. The tumor may grow rapidly after birth, but unilateral hydronephrosis often escapes notice for many months. Bilateral hydronephrosis is associated with fatal anuria.

(2) The **acquired form** often causes no noticeable symptoms. Or there may be pain, aching, and a dragging sensation in the kidney region. These vary in intensity and persistence, and are most marked when the tumor is growing rapidly. A tumor develops in the lumbar and iliac region. It is seen and felt to be rounded, sometimes lobulated, not movable (except with movable kidney), tense, and is flat on percussion. In front and adherent to it may be seen or felt a sausage-shaped mass, the colon, that is tympanitic, at least after inflation by rectum. The rate of growth may vary from time to time, the tumor sometimes grows smaller or even disappears.

Intermittent Hydronephrosis.—The tumor grows rapidly, the pain may be marked and the urine lessened. Then there is marked increase of urine, which may be clear or turbid, and the tumor becomes smaller or disappears. After a time this cycle may be repeated. This is usually due to temporary obstruction of the ureter with a movable kidney, and the tumor may be movable.

The cyst may be infected, and pyonephrosis results, with symptoms of sepsis. In uninfected cases the general health is often quite good. In bilateral cases anuria is common. The cyst rarely ruptures. Recovery after emptying of the cyst may occur. The diagnosis may be helped by aspiration or by cystoscopy and ureteral catheterization.

Treatment.—With movable kidney a pad and binder may relieve. Massage, possibly, may empty some pelves. Surgery may be indicated to remove the obstructing stone, etc., or to empty or remove the tumor.

QUESTIONS.

Give the etiology, lesions, symptoms, and treatment of hydronephrosis.

Discuss intermittent hydronephrosis.

PYELITIS.

Etiology.—Usually bacterial with streptococci or staphylococci of suppuration, typhoid bacilli, tubercle bacilli, pneumococcus, etc. The infection may reach the pelvis from the lower urinary passages, especially with urinary stasis; from acute or chronic inflammations of the kidney, cancer, parasites, tuberculosis; from the perirenal tissue or peritoneum. Pyelitis may occur in infectious diseases—typhoid, small-pox, scarlatina, etc. It may start in the pelvis—tuberculosis, cancer, parasites, trauma, exposure to cold, renal stone. It may be due to toxic substances not bacterial, as cantharides, etc., or in diabetes.

Lesions.—They vary from the mildest catarrh to suppuration and necrosis. Mild cases show congestion, swelling, cloudiness, increased secretion, and sometimes ecchymoses of the wall. There may, in severer forms, be suppuration or a fibrinous or necrotic false membrane. The contents of the pelvis are turbid from desquamated epithelium, or may be purulent, thick pus, or almost pure blood. In protracted cases the walls may be thickened or dilated. The ureters are usually inflamed and may be occluded. The kidney is often involved, showing acute nephritis, chronic nephritis, or small abscesses (suppurative pyelonephritis) that may or may not open into the pelvis; or the kidney and pelvis may together form one large abscess cavity (pyonephrosis), with more or less dilatation and necrosis of the kidney. The collections of pus may be partly absorbed, leaving putty-like masses that may become calcified—this is quite common in tuberculosis. The lesion may be bilateral or unilateral.

Symptoms.—**The Urine.**—In the milder cases it may contain bacteria, transitional epithelium from the pelvis. In severer cases it contains pus, blood, and granular or cheesy matter. It is usually acid, but there may be ammoniacal fermentation in some cases. The pus alone may give a small amount of albumin in the filtered urine, but there is very often an associated nephritis that gives albumin and casts. **Other symptoms** are not always present, but sometimes the pyelitis seems to cause pain and deep tenderness over the affected kidney. There are sometimes attacks of chills, fever, and sweating, during or at the end of which the pain and pyuria become more marked. Such attacks may last a few hours or several days. They may recur at longer or shorter intervals, at times quite regularly, suggesting intermittent malaria. The general condition varies with the degree of sepsis. With ammoniacal urine and some obstruction there may be an attack of dyspnea, restlessness, stupor, coma and death (ammonemia), suggesting diabetic coma. Some cases give the signs of pyonephrosis. The mild cases, such as those with typhoid, usually recover completely. Chronic suppuration persists, and the patient is apt to die of sepsis or the accompanying nephritis.

The **diagnosis** may be helped by cystoscopy and ureteral catheterization. With tuberculosis the bacilli are in the urine. Many cases can not be positively diagnosed, except at autopsy.

Treatment.—Attack the cause when feasible. For the pyelitis, rest in bed during exacerbations, hot applications to loins for the pain. The patient should drink much water. Urotropin, turpentine, etc., may perhaps help. Surgical treatment may be indicated.

QUESTION.

Give the etiology, lesions, symptoms, and treatment of pyelitis.

CONGENITAL CYSTIC KIDNEY.

Lesions.—The kidney is represented by a large lobulated mass that may weigh two or more pounds. It is a mass of *cysts, the wall* may contain some flattened kidney tissue. It is lined with flat epithelium. The contents are a yellowish or brown liquid containing albumin, cholesterin, crystals derived from hemoglobin, but usually no urea or urates. Often both kidneys are affected.

Symptoms.—Labor may be obstructed. The tumors often cause dyspnea and cyanosis by pressure on the lungs. The child usually dies within a few days. In some cases the tumor is small at birth, and may not be discovered until it begins to increase rather rapidly. This may be in adult life. There are the symptoms and physical signs of a lobulated tumor, tense and elastic, on one or both sides, together with the symptoms of chronic interstitial nephritis. The patients do not live more than a year or two after the enlargement becomes marked. They die of the nephritis.

The **treatment** is that of the nephritis. If the other kidney is quite normal, a cystic kidney may be excised.

QUESTION.

Give the lesions, symptoms, and treatment of congenital cystic kidney.

MALIGNANT NEW GROWTHS.

Primary growths usually are sarcoma, commonly in children up to five years of age; or medullary carcinoma, usually in children up to five or in adults past fifty years. The sarcoma may contain striped muscle. Other forms of carcinoma may occur. Secondary tumors are not rare. The primary tumors generally involve the whole of one kidney and may become very large, the sarcomata sometimes weighing many pounds. Secondary tumors may be in parts of both kidneys.

Symptoms.—Cancerous cachexia, and a tumor, visible and felt in the lumbar region. It may become enormous; is not very hard, often lobulated, flat on percussion, but the colon lies over it, and usually it is not movable. **Urine**: Often albuminous, contains much or little blood at times, and possibly multinucleated cells. The disease is usually fatal in a year or two.

Treatment is surgical.

QUESTION.

Give varieties, causes, symptoms, and treatment of new growths of the kidney.

PERINEPHRITIC ABSCESS.

Etiology.—Caused by streptococcus or staphylococcus pyogenes, colon bacillus, typhoid bacillus, or pneumococcus. It may extend (*a*) from the kidney, its pelvis, or ureter in pyelonephritis, cancer, or tuberculosis; (*b*) from perforation of the appendix or colon; (*c*) from spinal caries, inflammations of pleura, etc.; (*d*) it may follow trauma, either a contusion or a wound of the perirenal tissue; (*e*) may occur in infectious fevers. It is usually in adult males.

Lesions.—The loose cellular tissue around the kidney is the seat of a cellulitis, but later a distinct abscess cavity may be formed. The inflammation begins behind the kidney, but may completely surround it or be found in front only. The pus burrows in every direction and may rupture. The kidney is often included in the suppuration.

Symptoms.—The onset occasionally is abrupt with a chill, but usually more gradual. The general symptoms are those of sepsis. Locally there are pain and tenderness at the site of the kidney. The pain may radiate along the course of the ureter and down the thigh or around the flank. *Examination:* The spine is flexed laterally, concave toward the affected side, and is rigid. The thigh is partly flexed, the trunk leans forward in walking. The skin in the loin and flank may be red or edematous and swollen. On palpation the tissues feel thickened and firm over the kidney, but the induration is not sharply defined. Tenderness is present. Later there may be a rather distinct tumor, that possibly may fluctuate. It is dull on percussion, but the colon may be made out over it. The abscess may rupture externally, leaving a persistent sinus; or into the lung, or a hollow abdominal viscus with formation of a fistula and escape of pus; or into the peritoneum with fatal peritonitis; or into the pleura with empyema. The pus is very often extremely foul. The course may be acute at the start, but may become chronic. Amyloid disease may follow. Spontaneous recovery is very rare. The disease may last from two weeks to a few years.

Treatment is purely surgical.

QUESTION.

Give the etiology, lesions, symptoms, and treatment of perinephritic abscess.

DISEASES OF THE NERVOUS SYSTEM.

GENERAL CONSIDERATION.

Motor Tract.—This extends from the motor area of the cortex to the motor end-plates on the muscle-fibers. It consists of two sets of neurons or segments.

Upper Segment.—The cell bodies are in the motor area of the cortex, the axons end around the cells of the nuclei of the motor cranial nerves and the anterior horn cells of the spinal column. The *lower segment:* The cells are in the nuclei of the motor cranial nerves and the anterior horn of the spinal column. The axons end in the motor end-plates.

Upper Segment.—*The Motor Area of the Cortex.*—In the cerebrum these cells are grouped around the fissure of Rolando, in the frontal and parietal lobes and the paracental lobule. The center for the leg is in the mesial portion and the contiguous part of the convexity; below this are the centers for the arm and hand; somewhat more anteriorly, those for the head and eyes; the lowest are those for the face and mouth. The axons (the pyramidal fibers) run from the cells in the cortex, in the corona radiata, converge to the internal capsule, occupying the genu and the anterior two-thirds of the posterior limb, the fibers for the face being in front, and those for the lowest spinal centers most posterior; then they enter the cerebral peduncle, those for the face being mesial, for the lower spinal centers lateral. Here some fibers cross to the other side to the opposite third nerve nucleus. The remaining fibers pass into the pons, having given off the fibers destined for the opposite fourth, fifth (motor nucleus), and sixth cranial nuclei. In the posterior half of the pons the fibers for the seventh nucleus on the opposite side branch off, the remainder go down into the medulla, send off decussating fibers to the ninth, tenth, eleventh, and twelfth motor nuclei. At the lower end of the medulla most of the remaining fibers cross to the other side of the middle line in the decussation of the pyramids, and run down the cord in the lateral pyramidal column, giving off filaments to the groups of anterior horn cells in every spinal segment. The smaller part of the bundle forms the direct pyramidal column, and gradually decussate in the anterior commissure and end around the anterior horn cells. The axons end in arborizations around the cells of the motor nuclei. It will be noted that the axons from the cells of the facial area of the cortex end around the cells of the cranial motor nerves to the face, the cells of the arm area send their axons to the cervical area of the cord, from the leg area the axons go to the lumbar and sacral parts of the cord; that all fibers cross the middle line to nuclei on the opposite side and the bundles of pyramidal fibers gradually fade away.

Lower Segment.—The cell bodies are in the nuclei of the cranial motor nerves and in the anterior horn cells of the spinal cord. The axons run in the motor cranial nerves and in the anterior spinal nerve-roots and spinal nerves, and terminate in motor end-plates. These do not decussate.

The cortical cells have to do with voluntary motion, and they seem to exercise a constant effect on muscular tone, probably an inhibition. The cells of the lower segment direct the impulses resulting in motion and the nutrition of the muscle, and tend to maintain muscular tone.

Degenerations.—If a cell body is killed its axon is killed. If the axon be injured, the part distal to the injury will die. In the motor tract such degeneration is distal to the point of injury and the degeneration is called "descending." This degeneration and death is limited to the neuron injured. If the lesion is in the upper segment only, the lower segment remains intact and vice versa. If the muscle is deprived of impulses from the cell of the lower segment due to disease or death of the whole cell or a part of its axon, the muscle is paralyzed and flaccid; it atrophies, shows reaction of degeneration, and no reflexes. If the lower segment is uninjured, but be cut off from the cortex by disease of the pyramidal fiber or of the cortical cell, the muscle is paralyzed, but stiff and spastic—that is, the tone is increased; there is but slight atrophy, due only to disuse; the reflexes are exaggerated, there is no change in electric reaction.

A lesion may be (*a*) *irritative*, causing tonic or clonic spasm and perhaps increased reflexes. In the motor tract irritative lesions are usually in the upper segment and in the cortex. (*b*) A *destructive* lesion in the motor tract causes *paralysis*, a loss of power of voluntary motion. Paralysis may also be due to a lesion of the muscle itself. A moderate loss of power is sometimes called *paresis*. *Hemiplegia* is paralysis of one side of the body; *paraplegia*, of both lower extremiites with or without part of the trunk; *monoplegia*, of one limb.

Spasm is excessive involuntary muscular contraction; it may occur with paralysis. *Tonic spasm* is a long-continued tetanic contraction; it may last from a few seconds to many years. If protracted there is permanent shortening of the muscles, called *contracture*. *Clonic spasm* is a series of intermittent contractions often of considerable amplitude. *Tremor* is clonic spasm of very slight amplitude. *Convulsions* are sudden, violent spasms, clonic, tonic, or both, which may be localized in a few muscles, or one side of the body, or may be general.

Sensory Tract.—This is more complex and less understood than the motor tract. The cell body of the *most distal neuron* is in the ganglion on the posterior spinal nerve root or its homologues among the cranial ganglia. The axon from the cell in the posterior spinal ganglion branches like the letter T. One branch runs distally in the posterior root and the spinal nerve to various sensory endings; it is probably a dendrite. The other branch, representing the axon, runs centrally into the cord; here it branches, one part going down in the *comma tract*, gives off perpendicular branches (collaterals), and soon ends. The ascending branches seem to follow various routes; the largest bundles of these fibers make up the *posterior columns of Burdach and Goll*. All seem to give off collaterals. The collaterals end in arborizations around (*a*) the anterior horn cells; (*b*) the cells of Clarke's column (whose axons form the direct cerebellar tract); and (*c*) around other cells whose axons cross the middle line and form the anterolateral ascending tract of Gowers and the ground-bundles. The axons of the posterior columns arborize around cells in the nucleus gracilis and the nucleus cuneatus in the posterior part of the medulla. *Neurons of the second order:* The cells of Clarke's column and some other spinal-cord cells are in this class. Also the cells from the slender and cuneate nuclei, whose axons go upward, cross to the other side in the decussation of the fillet, run in the fillet and the tegmentum, and end in arborizations around the cells of the optic thalamus. The cells of the optic thalamus (*third order*) send their axons to the cerebral cortex to arborize around the cortical cells of the motor area and other parts of the parietal lobe. There are many other routes that are less understood, some axons soon cross to the other side of the cord and ascend there. In the internal capsule sensory fibers occupy the posterior part. In the cortex the sensory area corresponds to the motor area and other parts of the parietal lobe. Various sensations travel by different routes, but very little is known about them.

All parts of the body have sensory nerves, but for topical diagnosis the *cutaneous sensations* are most important. There are several subdivisions of sensation. There is the sense of *contact* (pure tactile sensation), of *pressure*, of *localization* of the site of the stimulus and area of contact, of *temperature* (all these are grouped as *tactile sensations* by some authors); also *pain*, *electric sensibility* and such sensations as *tickling*. These are sometimes called *common sensibility*. Muscular sense, stereognostic sense, etc., concern the deeper tissues.

Anesthesia is a loss of sensibility. **Hyperesthesia** is abnormal acuteness of sensibility. **Paresthesia** is disordered sensibility, such as formication, tingling, numbness, etc. Sensation may be *delayed* (even ten or fifteen seconds). The delay may affect all sensation or only one kind. For instance, touch may be normal, but the sensation of pain delayed.

Reflex Action.—The reflex are for muscular reflexes includes the muscle, the end-plates, the entire neuron of the anterior horn, the sensory neuron of the posterior spinal ganglion and the sensory end-organ. Loss of function at any point results in loss of reflex. Exaggerated function at any point may result in increased reflex. There are very many motor reflexes (see p. 356): (1) **Tendon or Deep Reflexes.**—*The knee-jerk:* When the knee is partly flexed and the muscles relaxed, a blow on the tendon below the patella causes a sudden contraction of the quadriceps extensor with extension of the leg. This is present in health; it may be exaggerated or lost in disease. The motor neurons are in the second lumbar segment. The sensory nerves are those ending in the muscle, and have to do with myotatic irritability. *Ankle clonus* is absent in health, present in certain abnormal states. If the ankle be suddenly flexed and the foot held in flexion, there results a clonic spasm of the muscles of the tendo Achillis. The motor center is in the fifth lumbar segment. The *triceps, posterior* and *anterior wrist* tendon reflexes are also important. (2) **Superficial or Cutaneous Reflexes.**—The sensory nerves end in the skin. Very many are mentioned in the table. *The plantar reflex:* Normally, upon tickling the sole all the toes are flexed. The *Babinski reflex* (extensor plantar reflex) is a pathologic modification of this, in which at least the great toe is extended. It is found with loss of function of the upper neuron. (3) **Organic reflexes** are connected with micturition, defecation, deglutition, etc.

Nutrition is often disturbed in nervous diseases. **Nutrition of the muscle** depends upon the cells in the anterior horn or the nucleus of the cranial motor nerve—that is, upon the lower segment; with loss of function of these neurons, the muscle shows lower segment paralysis—very marked wasting, atrophy, loss of tone, is flabby, and gives the reaction of degeneration. With these neurons intact, if the muscle is not used for any reason, there may be slight wasting, but no reaction of degeneration. The *nutrition of the skin and joints,* etc., is controlled by fibers running with the sensory nerves.

Electrical Reaction.—Normally, the muscle contracts (*a*) when the nerve only is stimulated (indirect stimulation), or (*b*) upon stimulation of the muscle itself (direct stimulation) by either the galvanic or faradic current. The **galvanic current** causes a single, quick contraction either upon the make or upon the break of the circuit. A sudden increase in the strength of the current is equivalent to closing a circuit; a sudden decrease is equivalent to breaking the circuit. If with the galvanic current the kathode is nearer the muscle than the anode it is called a *descending current.* If the anode is nearer than the kathode it is an *ascending current.* In stimulating the nerve neither electrode is over the muscle, but some distance from it. In direct stimulation of the muscle one electrode is over the muscle, the current affects the muscle directly, and also the end-plates and terminal filaments of the nerve. By varying the strength and direction of the current, it is found in both direct and indirect stimulation that the weakest galvanic current that will cause a contraction of the muscle is a descending current, and that the contraction occurs when the circuit is closed. This is called the kathodic (or cathodic) closure contraction, abbreviated K.C.C. (or C.C.C.). Ascending currents that are somewhat stronger cause a contraction at the closure of the circuit, called the anodic closure contraction, A.C.C. Also with a current of about the same strength when the circuit is broken, an anodic opening contraction, A.O.C., occurs. The relation between the anodic and kathodic closure contractions in health is expressed therefore by the formula K.C.C. > A.C.C. The contraction at the opening of the descending current (K.O.C.) and of the ascending current (A.O.C.) are less important. **Faradic stimulation** of either muscle or nerve causes a tetanus as long as the current lasts or until the muscle gradually relaxes from fatigue.

The **reaction of degeneration** (R.D.) is present when the muscle is completely cut off from the neurons of the cord, but two or three weeks are needed for its complete development. In this condition the *nerve* can be stimulated neither by faradism nor galvanism. The *muscle* can not be stimulated by faradism, but it is abnormally sensitive to galvanic currents. A much weaker current than in health causes a contraction, also the weakest effective current is ascending with the contraction at closure (A.C.C.). To get a contraction at the closure of a descending current a much stronger current is necessary. This is expressed by the formula A.C.C. > K.C.C. The contraction is not a quick jerk, but develops slowly, and relaxes slowly, also mechanical irritability of the muscle is increased.

Often a *partial reaction of degeneration* is found. Usually the sensibility of the *nerve* to both faradism and galvanism is lessened or lost. The *muscle* may react to strong faradic currents; and with galvanic currents, usually K.C.C. = A.C.C. or A.C.C. > K.C.C. When the reaction of degeneration has developed the muscle gradually atrophies, the fibers degenerate, and may be replaced by connective tissue. In time such muscles may lose all electric excitability. Or they may recover in the course of many weeks. During recovery there is a reversal of the changes of electric activity: first, a partial reaction of degeneration and, finally, normal reaction. In recovery, voluntary motion returns before normal electric reactions.

QUESTIONS.

Describe the upper segment of the motor tract.
Describe the lower segment of the motor tract.
Describe secondary degeneration in the motor tract.
What are the irritative symptoms of the motor tract?
What are the types of paralysis?
Discuss spasm.
Discuss the sensory tract.
Give the chief varieties of sensation.
What are the irritative symptoms?
What are the paralytic symptoms?
Discuss deep and cutaneous reflexes.
What neurons preside over trophic symptoms in muscles?
What is the course of trophic impulses to the skin and joints?
Discuss the normal electric reactions in nerve and muscle.
Describe the reaction of degeneration and the associated state of the muscle.
In which motor segment is the lesion?
What is a partial reaction of degeneration?

DISEASES OF THE NERVES.

NEURITIS.

Etiology.—Focal or Localized.—It follows (*a*) exposure; (*b*) extension from contiguous inflammation; (*c*) trauma—wounds, pressure, stretching of nerves, etc.; (*d*) perhaps is sometimes an infection, or (*e*) due to a chemic poison.

Lesions.—The lesion may develop acutely or chronically. It may involve a small part of the nerve (*focal*) or a considerable part of the nerve (*diffuse or disseminated*). A lesion at first focal may become diffuse (*migratory*). A *localized neuritis* is limited to one nerve; *multiple neuritis* attacks several nerves in different parts of the body. (1) *Parenchymatous neuritis:* The nuclei of the sheath of Schwann are increased in number, the cell protoplasm is more abundant.

The myelin segment collects in drops and disappears. The axis cylinder becomes granular, broken, and disappears. The same changes develop in that part of the axon distal to the original lesion. This is called *secondary* or *Wallerian degeneration.* Such a secondary degeneration occurs whenever an axon is cut off from its nerve cell. That part of the neuron above the lesion does not degenerate. The rest of the nerve structure is not changed. (2) *Perineuritis* and *interstitial neuritis*—Acute: The nerve is red, swollen, and ecchymotic. Microscopically, the vessels are congested; there is exudative and acute productive inflammation of the connective tissue around the nerve in *perineuritis*, and throughout the nerve in *interstitial neuritis.* The nerve-fibers often are but little changed, but sometimes they show marked degeneration (*mixed neuritis*). In focal neuritis the lesion is often near joints and at a bend of the nerve. Chronic: This shows a chronic productive inflammation of the connective tissue of the nerve. It is often dark from old hemorrhage. The nerve-fibers may be unaffected.

Symptoms.—The constitutional symptoms are slight or absent. The onset may be abrupt or gradual. At first irritative symptoms are apt to appear, later those of paralysis. **Sensory symptoms** (in a sensory or a mixed nerve): At first there may be hyperesthesia and hyperalgesia; pain in the nerve and its distribution, often boring and severe; increased upon pressure or upon stretching the nerve; paresthesia—numbness, formication, tingling, etc.; and later, in severe cases, loss of sensation. **Motor symptoms** (in a motor or a mixed nerve): At first twitching and rigidity of the muscles, later weakness and paralysis. The wasting is very marked. The nerve and muscles are tender. The reaction of degeneration gradually develops. In the worst cases contractures may develop in time. **Tendon reflexes** at first may be increased, later are diminished or lost.

Vasomotor and Trophic Disturbances.—Pallor and cyanosis; coldness of the skin; hyperidrosis, anidrosis; the skin may be red and shiny, or edematous, or thickened or scaly. The hair may be coarse and brittle, the nails thick and rough. There may be herpes or swelling of the joints.

Course.—The onset may be *acute*, the course may be short, and recovery within two to four weeks may be perfect. Other cases drag on, there is a loss of function for many weeks, but, finally, recovery may be quite complete. Or the onset may be *insidious* and gradual, the severity slight or severe, the duration short or very long. The neuritis may successively invade nerves nearer the head or the cranial nerves also (ascending neuritis).

Electric Reaction and Prognosis.—If there is *no change* in the electric reactions, the prognosis is very good and recovery takes place within two or three weeks. With *complete R. D.*, recovery may not occur from two to fifteen months and perhaps may never be perfect. With *partial reaction of degeneration*, the prognosis is good, but the duration of the symptoms may be several weeks. Voluntary motion returns before normal electric reactions. Sensory functions are often restored early.

Treatment.—The cause should be removed or treated when possible. In the acute stage use local rest and anodynes, such as hot fomentation or galvanic electricity. After the acute stage, strychnin and other tonics; for the muscles massage, galvanism, passive movements, and warm salt or sulphur baths.

MULTIPLE NEURITIS OR POLYNEURITIS.

Etiology.—Due (1) to the poisons of infectious diseases—leprosy, beriberi, diphtheria, typhoid, pneumonia, variola, tuberculosis, syphilis, septic diseases, etc. (probably herpes zoster belongs here). (2) To such poisons as alcohol, ether, carbonbisulphid, naphtha, lead, arsenic, mercury. (3) An auto-intoxication in gout or diabetes. (4) Cachexia in anemia, cancer, tuberculosis—perhaps these are also auto-intoxication. (5) Without obvious cause, or after exposure to cold—probably a primary infection.

Lesions.—Like those of local neuritis, but in several nerves in different parts of the body. Often symmetric and in the distal part of the nerve.

Symptoms.—There are many types.

(1). **Acute febrile polyneuritis** may occur spontaneously or after exposure. The onset is abrupt with a chill, fever (104° F.), and all its symptoms, with marked pain in the back and limbs, often with delirium. There are the symptoms of neuritis—motor, sensory, trophic, etc.—often appearing first in the legs, spreading upward, but sometimes occurring first in the arms. *Foot-drop* is due to paralysis of the flexors of the foot, and results in the "steppage" gait. *Wrist-drop* is due to paralysis of the extensors of the wrist. The trunk and face are sometimes involved. Occasionally the sensory and trophic symptoms are but slight. *The bladder and rectum are rarely affected.* The *worst cases* die in from one to two weeks from paralysis of the respiratory muscles or the heart, or in coma. The *moderate cases* get worse for from five to six weeks, then remain stationary for a while, and recover slowly. The *long cases* last a year or more and may have some contractures, but most of them recover in time.

(2) **Recurring Multiple Neuritis.**—A series of repeated attacks in the same nerves at long or short intervals.

(3) In **infectious diseases and cachectic states** the limbs are often involved and recovery follows. (See also Diphtheria, p. 72.)

(4) **Alcoholic multiple neuritis** occurs in steady drinkers, especially women. The onset is gradual. There often are sensory symptoms for weeks or months—neuralgia, paresthesia first below the knee, afterward in the forearm and hand; later motor symptoms appear, possibly irritative symptoms at first, afterward there is paralysis with wasting, R. D., and loss of deep reflexes, but the skin reflexes usually persist. There are foot-drop, steppage gait, wrist-drop, etc. Sometimes it involves the muscles of the thigh and upper arm also, or even the face and sphincters. The sensory symptoms vary extremely, as do the trophic symptoms, but edema is common. The *mental symptoms* are very striking; there are delirium, hallucinations, a loss of sense of time and place, and long imaginary journeys. The course is often protracted, recovery usually follows.

(5) **Lead-poisoning** (see also p. 132).—After long exposure to lead it develops, and usually there is the lead line on the gum, with colic and anemia. The most characteristic form has a bilateral musculospiral paralysis with wrist-drop, flexed fingers and adducted thumb, and the sensory and trophic symptoms of such a neuritis. The supinators regu-

larly escape. *Prognosis:* The disease is seldom fatal, but recovery is slow and often incomplete, because the patient usually begins again to absorb lead.

(6) **Arsenic neuritis** usually follows acute arsenic-poisoning. It resembles alcoholic neuritis without the head symptoms, and herpes is common. **Mercury** causes similar symptoms, but no herpes, and a tremor is present. **Beriberi** is an acute infectious disease (see p. 96).

Treatment.—As in other neuritis. An air-bed may be necessary to prevent bedsores. The elimination of the metallic poisons may be aided by potassium iodid, 30 gr. daily. In alcoholic cases, taper off the alcohol.

NEUROMA.

Nodular enlargements of the nerve may be **true neuromata,** composed of nerve-fibers (very rare); or **false neuromata,** composed of fibrous tissue, gumma, a leprosy nodule, sarcoma, etc. There may be hundreds of nodules scattered along the nerves throughout the body. **Amputation neuroma** is a nodule of fibrous and nervous tissue which develops on the stump of a nerve after amputation. Neuromata often cause no symptoms. Sometimes they cause pain, paresthesia anesthesia, spasm, paralysis, etc. If troublesome and accessible, the nodules may be excised.

QUESTIONS.

Give the etiology, lesions, and symptoms of localized neuritis.

Discuss the electric reactions and prognosis.

Give the treatment.

Give the etiology of multiple neuritis, the different varieties and their symptoms.

Discuss neuromata.

CRANIAL NERVES.

(I.) OLFACTORY NERVE AND TRACT.

Hyperosmia, undue keenness of smell, is found in hysteria and insanity. **Anosmia,** loss of smell, is due usually to nasal catarrh with a dry mucous membrane; less often to a destructive lesion in any part of the olfactory tract, from fractures of the cribriform plate, meningitis, brain tumors; or congenital absence of the center or nerves. **Parosmia,** perverted sensation of smell, or **hallucinations of smell,** may occur in the insane, as an epileptic aura, and with disease of the centers. The smell may be tested with oil of cloves or cinnamon, but not with such irritating substances as ammonia. Ordinarily the prognosis is bad.

QUESTION.

What are the disturbances of this tract?

(II.) OPTIC NERVE AND TRACT.

The Retina.—Albuminuric retinitis occurs in chronic (especially interstitial) nephritis, and with arteriosclerosis. The ophthalmoscope may show white patches around the macula lutea or elsewhere; or flame-shaped hemorrhages; or swelling of the disk from inflammatory effusion. *Retinitis* may occur also with syphilis, anemia, leukemia, diabetes, and hemorrhagic conditions.

Amaurosis is blindness without obvious lesion of the eye. **Amblyopia** is defective vision without obvious lesion in the eye. *Toxic amaurosis* may be due to lead, alcohol, perhaps quinin, often to uremia with or without convulsions. Usually it is transient.

Toxic amblyopia is usually from tobacco. It has a gradual onset, is bilateral, and most marked in the center of the visual field; there is always a central scotoma for red and green. It is probably a chronic neuritis starting in the fibers to the macula. Amblyopia or amaurosis may be hysterical.

Nyctalopia, or night-blindness: Vision is good in a bright light and defective in a dim light.

Hemeralopia, day blindness: Vision is good in a dim light, defective in a bright light.

The Optic Nerve and Tract.—The optic nerve runs from the retina to the optic chiasm. Here the mesial half of each optic nerve crosses in the chiasm to the other side; the lateral half continues on the same side. Fibers from each macula run to both optic tracts. The optic tract extends from the chiasm to the nerve centers. The retinal fibers in the optic tract go to ganglion cells (1) in the pulvinar (optic thalamus), (2) in the external geniculate body, and (3) in the anterior quadrigeminate body. From these ganglia fibers go in the posterior part of the internal capsule and the optic radiations to the visual perception center in the cortex of the occipital lobe (cuneus). The right halves of the visual field correspond to the left halves of the retinæ, and fibers from here from both eyes run in the left optic tract to the left half of the brain and vice versâ. **Localization of Lesions.**—A lesion of one optic nerve may affect all of the retina of that eye. A lesion of the middle part of the chiasm affects the nasal halves of both retinæ, consequently the temporal halves of both visual fields (*temporal hemianopia*). A lesion of the right lateral margin of the chiasm involving only the uncrossed fibers affects the temporal half of the right retina, causing *right nasal hemianopia.* If it affects the left margin of the chiasm it causes *left nasal hemianopia.* If it affects both right and left lateral margins, but not the middle of the chiasm, it causes *bilateral nasal hemianopia*, which is rare. This is called *heteronymous hemianopia.* A lesion in the right optic tract, thalamus, or occipital lobe affects the right halves of both retinæ, the left halves of both visual fields (*left homonomous hemianopia*), etc. The maculæ having fibers in both tracts escape, unless the lesion involves both tracts or both sides of the brain. Hemianopia may affect just half of each retina or smaller areas, which, however, correspond in both eyes.

The pupillary light-reflex arc includes the retina, optic nerve and tract, the primary optic ganglion cell, fibers from this to the third nerve nucleus; this nerve, the ciliary ganglion, the ciliary nerves, and the sphincter muscle of the iris. Lesions in any part of this arc destroy this reflex. Lesions back of this in the optic radiations or in the cortex cause hemianopia, but a ray of light on the blind part of the retina will cause reflex pupillary contraction; this phenomenon is called the *hemiopic pupil reaction.*

There may be hemianopia for color only and not for white light (*hemiachromatopia*). Lesions of both occipital lobes cause double lateral hemianopia, perhaps with complete blindness. In hemianopia the opthalmoscopic examination is negative. Hemianopia may be functional, in hysteria or migraine; usually it is due to organic brain disease, such as softening, hemorrhage or tumor; often hemiplegia and sometimes hemianesthesia are associated. If due to pressure, without destruction of nerve tissue, it may disappear.

Optic neuritis (*choked disk, papillitis*) is found with meningitis, brain tumor and abscess, Bright's disease, lead-poisoning, anemia, after the infectious diseases, or without discoverable cause. It is usually bilateral, the ophthalmoscope shows (*a*) congestion of the disk and blurring of its edges; or (*b*) deeper congestion, dilated veins, swelling and striation of the disk, and often hemorrhage. Recovery is sometimes complete or optic atrophy may follow. The retina also is often affected (neuroretinitis). The lesions may be far advanced before sight fails.

Optic Atrophy.—(*a*) *Primary*, found in tabes dorsalis, general paralysis, diabetes, and toxemia; there is also a hereditary form. The ophthalmoscope shows the disk to be gray, the edge well defined, the arteries normal. (*b*) *Secondary*, follows optic neuritis, various cerebral diseases, and pressure on the chiasm or optic nerves. The ophthalmoscope shows a very white disk with small arteries. The *symptoms* are dimness of vision, altered visual field, and affected color sense.

QUESTIONS.

Define the following terms: Albuminuric retinitis, amaurosis, amblyopia, nyctalopia, hemeralopia, temporal hemianopia, nasal hemianopia, homonomous hemianopia, heteronymous hemianopia, the pupillary light reflex, the hemiopic pupillary reaction.

Describe the optic tract.

Give the causes and symptoms of hemianopia, optic neuritis, optic atrophy.

(III. IV. VI.) THE MOTOR NERVES OF THE EYE.

The third nerve nucleus lies below the floor of the aqueduct of Sylvius. Its fibers pass through the crusta. After emerging, the nerve runs along the cavernus sinus through the sphenoid fissure into the orbit. It is the motor nerve for the levator palpebræ superioris, the superior, internal and inferior recti and the inferior oblique muscles; also of the constrictor of the iris and the ciliary muscle.

Irritation causes (*a*) **spasm** (in hysteria, hypermetropia, meningitis), usually affecting the levator palpebræ and the internal rectus. (*b*) **Nystagmus** is a rhythmic, clonic, bilateral movement of the eyeballs found in coal-miners, albinos, congenital cases, etc. (*c*) **Myosis** may be due to stimulation of the sphincter of the iris, or to the paralysis of the sympathetic.

Paralysis.—Paralysis of the levator palpebræ (ptosis) (1) is often congenital and incurable; or (2) due to a lesion of the third nerve (*a*) in its superior division, associated with paralysis of the superior rectus; or (*b*) of the whole nerve or its nucleus, with paralysis of all the muscles supplied by the nerve. (3) Cerebral lesions may cause ptosis without affecting any other branch of the third nerve. (4) There may be hysteric cases. (5) Pseudoptosis is due to a sympathetic nerve lesion with vasomotor paralysis and myosis. (6) Ptosis is found in idiopathic muscular atrophy that attacks the face muscles also.

The **third nerve trunk** may be involved in meningitis, gumma, aneurysm, in neuritis as in diphtheria, or tabes dorsalis. Lesion of this nerve causes ptosis, paralysis of all the external muscles of the eye (except the superior oblique and the external rectus) with diverging strabismus and diplopia, mydriasis, no pupillary light reflex, and loss of accommodation.

The paralysis may involve only the branches to the internal eye muscles, or to the levator palpebræ and superior rectus.

Cycloplegia, paralysis of the ciliary muscles, causes loss of power of accommodation. Distant objects look distinct; near objects look blurred, but a convex lens corrects this. It is found in diphtheria and tabes.

Iridoplegia.—The pupils are usually (but not always) very small (spinal myosis). **Accommodative iridoplegia**: There is no pupillary contraction when the eye is focused on a near object. **Reflex iridoplegia**: A bright light suddenly brought before the eye causes no myosis. **Argyll-Robertson pupil**: There is loss of light reflex, but persistence of accommodative reflex. **Skin reflex iridoplegia**: Pinching or pricking the skin of the neck causes no dilatation of the pupil.

The **fourth nerve** supplies the superior oblique muscle which directs the eye downward and rotates it inward. This movement is defective in fourth-nerve paralysis.

The **sixth nerve** is rather long and therefore vulnerable. It innervates the external rectus muscle. It may be affected in meningitis, gumma or other tumors. Paralysis results in internal strabismus and diploplia on looking to the paralyzed side. The sixth-nerve **nucleus** seems to control the internal rectus of the other eye to a large extent (by fibers to the opposite third-nerve nucleus), therefore, in a lesion of the right sixth-nerve *nucleus* there is conjugate deviation of both eyes to the left, and the sixth-nerve nucleus is so near the seventh nucleus that there is usually right facial paralysis also.

General Features of Motor Paralysis of the Eye.—(1) There is **loss** of that movement for which the paralyzed muscle is responsible. (2) There is **strabismus** (squint)—*i. e.*, a lack of correspondence in the axes of the two eyes. *Divergent strabismus* is due to paralysis of the internal rectus; *convergent strabismus*, to that of the external rectus. With strabismus, if the *sound* eye be fixed on an object the *affected* eye will not look exactly at the object. The extent of this deviation varies with the degree of weakness of the muscle. This is called the **primary deviation.** (3) If the *affected eye* be fixed on the object the *sound eye* will be found to deviate more from the object than the bad eye did before. This deviation of the sound eye from the object is the **secondary deviation.** The habit of binocular vision leads to equivalent impulses being sent to the muscles that act together to move the eyes in harmony. If the strength of one of these muscles is lessened, the ordinary impulse sent to it causes less contraction, and therefore less motion in the eye, than is usual. This results in the primary deviation (when the sound eye is fixed upon the object). If now, the attention is centered on the weak eye, and strong enough impulses are sent to its weakened muscle to hold it in position, these stronger impulses, going to the strong eye also, result in too great contraction, and the sound eye is forced farther out of position, giving the secondary deviation (when the bad eye is fixed upon an object). (4) **Erroneous projection**: The abnormal effort of the weakened eye muscles in fixing the eye on objects leads to an inability to judge accurately of position. This confusion may lead to ocular vertigo. (5) **Double vision**: The images do not fall upon corresponding parts of both retinæ, because one or the other eye deviates; this results in the perception of two visual images; the one seen by the sound eye is called the true image; the one in the defective eye the false image.

Ophthalmoplegia is a chronic progressive process that may involve only the external muscles of the eye (**external ophthalmoplegia**), or only the internal muscles of the eye (**internal ophthalmoplegia**), or both external and internal muscles (**total ophthalmoplegia**). It may be due to lesions in the nuclei or elsewhere. The course is usually chronic.

Treatment of Ocular Paralysis.—The cause may be amenable to treatment. In acute cases hot fomentations, counterirritation or leeches to the temple may relieve the pain. *Diplopia* if slight may be treated with prisms, otherwise by placing an opaque glass in front of the injured eye.

QUESTIONS.

Give the symptoms of irritation of the optic nerves and its causes.

What is the definition and etiology of ptosis?

Define cycloplegia, Argyll-Robertson pupil.

Give the symptoms of paralysis of the third nerve, of the fourth nerve, of the sixth nerve, of ophthalmoplegia.

Define strabismus, primary deviation, secondary deviation, erroneous projection, double vision.

(V.) TRIGEMINAL NERVE.

The nuclei are in the floor of the fourth ventricle, in the pons and medulla, and extend downward into the cord. **Sensory fibers** pass through the Gasserian ganglion, and emerge in three trunks: (1) The ophthalmic nerve—the sensory nerve to the eyes, eyelids, frontal region, part of the nose. A branch goes through the supra-orbital foramen. (2) The superior maxillary nerve—the sensory nerve of the temple, cheek, nose, upper lip, nasal cavity, upper teeth, upper part of the mouth. It sends a branch through the infra-orbital foramen. (3) The inferior maxillary nerve—the sensory nerve for the lower teeth, lower part of the mouth, lower lip, the chin, part of the cheek, and the auriculotemporal region. It sends a branch through the inferior dental foramen. It also carries gustatory nerves: (*a*) branches which join the seventh nerve, go with it through part of the temporal bone, emerge as the chorda tympani, and are distributed to the anterior two-thirds of the tongue; also (*b*) other branches which join the glossopharyngeal nerve and reach the posterior third of the tongue and the soft palate. Note that the taste of the anterior two-thirds of the tongue may be destroyed by a lesion of the seventh nerve, that of the posterior one-third by a lesion of the glossopharyngeal nerve, and that all taste is destroyed by a lesion of the fifth nerve inside of the cranium. The **motor trunk** joins the inferior maxillary below the Gasserian ganglion and supplies the masseters, temporals, pterygoids, and some smaller muscles.

Lesions of the fifth nerve may be: (1) In the pons—hemorrhage, sclerosis or tumors. (2) At the base of the brain—meningitis, tumor, caries of the bone, etc.; these involve all branches. (3) Outside of the skull one or two branches may be involved in neuritis, tumors, aneurysm, wounds, etc. (4) Fracture of the skull may injure the nerve.

Symptoms.—Sensory Portion.—The symptoms are referred to the distribution of the entire nerve or some of its many branches (see p. 338). **Irritation** causes pain, often severe, tenderness along the nerve, hyperesthesia. **Loss of function** causes anesthesia and, as a result of this, slower muscular movements; loss of conjunctival reflex. **Trophic symptoms**: Secretion at first may be lessened with anosmia, dry mouth, defective taste; later, secretions may be increased in some cases. The gums become inflamed and ulcerated, the teeth loosen and decay; there may be conjunctivitis, corneal ulceration, perhaps with perforation and destruction of the eye (neuroparalytic ophthalmia), or herpes, especially supraorbital. **Gustatory nerves** may escape if the lesion is in the pons or distal to the Gasserian ganglion. They may be involved with a lesion in certain parts of the seventh nerve or ninth nerve. The paralysis of taste may be limited to the anterior two-thirds of the tongue or the posterior third.

Motor Symptoms.—*Irritative Symptoms.*—*Spasm* may be *tonic* (trismus) and often painful, in tetanus, hysteria, etc. Or *clonic*, in paralysis agitans; or without marked nerve lesion, especially in old women; or as chattering of the teeth in a chill, or gnashing of the teeth. In chorea, there may be single spasms of the muscles of the jaw. *Paralysis* is shown by flabbiness of the masseter and temporal muscles upon attempted contraction; reaction of degeneration and atrophy may follow.

Treatment.—The cause may be amenable to treatment. Treat the neutitis as usual (p. 330).

QUESTIONS.

Describe the trigeminal nerve.

Give the causes and symptoms of its sensory, gustatory, and motor disturbances.

(VII.) FACIAL NERVE.

The nucleus is in the floor of the fourth ventricle, near the nuclei of the sixth, eighth, and twelfth nerves. Its fibers run near those of the eighth nerve to the geniculate ganglion, go through the Fallopian aqueduct in the petrosal part of the temporal bone, and emerge through the stylohyoid foramen. It is the motor nerve for the muscles of expression, the orbicularis palpebrarum, stapedius, buccinator, and the muscles of the auricle. The taste fibers of the anterior two-thirds of the tongue join the seventh nerve at the geniculate ganglion, run with it nearly to the end of the Fallopian aqueduct, and then leave it as the chorda tympani. These points have diagnostic value.

Spasm occurs (*a*) especially in nervous persons, and those with neuropathic heredity; (*b*) it may follow pressure on the nerve-trunk or a lesion in the cortex; (*c*) is often reflex in eye-strain, trigeminal neuralgia, with diseased teeth or intestinal wounds; (*d*) in children a habit is often formed by imitation of similar spasm in others.

Blepharospasm is a tonic spasm resulting in the sudden closure of both eyes, which may last minutes or hours; or a clonic twitching of the lids (the most common form of facial spasm).

Associated with blepharospasm, other muscles of one or, less often, of both sides of the face twitch and jerk. This is more marked in fatigue and under excitement. Facial spasm is apt to persist throughout life with remissions and exacerbations. There may be tender points over branches of the fifth nerve.

Treatment.—Before the habit is too firmly established removal of the cause of irritation may result in a cure. Over the tender points, blisters, the thermocautery or an ascending galvanic current may be applied. General tonics may help.

Facial Paralysis (Bell's palsy).—(1) The lesion may be **above the nucleus** in the superior motor segment (the cortical neuron): (*a*) Rarely, a small lesion over the cortical motor face-area may cause facial monoplegia. (*b*) Usually these lesions are a part of a hemiplegia. The eyelids and forehead are not affected; but the other facial muscles on the side opposite to the lesion lose voluntary motion, but may retain some motion in facial expressions, such as smiling. Reflexes persist and may be exaggerated. The electric reactions are normal. There is no wasting. Contractures may develop.

(2) **Nuclear paralysis** may be due to (*a*) hemorrhage, tumor or softening at the nucleus in the pons. Usually the sixth-nerve nucleus also is injured, and often the pyramidal fibers, going down to the other side of the body. This results in a *crossed hemiplegia.* (*b*) The nucleus may degenerate, as in diphtheria, or, rarely, in anterior poliomyelitis.

(3) **Infranuclear Paralysis.**—The lesion may be (*a*) at the base of the brain—meningitis, tumors, aneurysm, hemorrhage; (*b*) in the Fallopian canal—a neuritis following cold or otitis media, or a fractured base; or (*c*) below the stylomastoid foramen—from injury, especially in a forceps delivery, etc.; (*d*) the facial nerve may be involved in multiple neuritis or syphilis.

Symptoms.—Slight irritation may first appear, pain, tenderness over the nerve, muscular twitchings, vertigo. The paralysis is almost always unilateral. If complete the affected side of the face is flat, expressionless, smooth, the lower eyelid droops, tears run over the cheeks. With attempted movements the forehead is not wrinkled, the eye can not be closed, the ala nasi is immobile; in whistling, the cheek bellies out; the lower lip droops; in smiling, snarling or laughing, the angle of the mouth is pulled toward the middle line, and the lips are flabby; in drinking, the liquid runs over the chin;

in eating, food accumulates in the affected cheek; labial sounds are imperfectly articulated. The nasal cavity is deprived of tears and is dry; and there is a defective sense of smell. The tongue really is protruded straight forward, but it seems to be deflected toward the paralyzed side. With stapedius paralysis there is hyperacusis in that ear. If the taste fibers are involved there is loss of taste in the anterior two-thirds of the tongue, and lessened saliva. Reflexes are lost, electric reaction is changed, wasting is marked. After many months contractures may develop in the paralyzed muscles, restoring somewhat the wrinkles and folds of the face, but attempted motion shows the paralysis.

Topical Diagnosis.—(1) If the lesion is below the stylomastoid foramen there is a pure motor paralysis of the face only; (2) if the lesion is in the very end of the Fallopian canal, paralysis of the auricular muscles also; (3) with a lesion just above the origin of the chorda tympani, there are loss of taste and lessened secretion of saliva also; (4) a lesion between the nerve for the stapedius and the geniculate ganglion causes hyperacusis also; (5) with a lesion above the geniculate ganglion or in the nucleus, all the above symptoms are present, except loss of taste and lessened saliva, and there is very apt to be involvement of the eighth nerve also. For the duration, prognosis, and electric reactions, see p. 330.

Treatment is that of a neuritis (see p. 330). The cause may be amenable to treatment.

Facial diplegia, bilateral facial paralysis, may rarely occur in bilateral lesions of the pons, in diphtheria, or in double otitis media.

QUESTIONS.

Describe the seventh nerve.
Discuss facial spasm.
Give the symptoms of upper segment paralysis.
Give the symptoms of lower segment paralysis.
How is the site of the lesion determined?

(VIII.) AUDITORY NERVE.

This nerve consists of two parts, cochlear and vestibular, which have different functions.

(1) The **cochlear nerve** supplies the organ of Corti in the labyrinth. The nucleus is in the medulla; the cortical center is in the middle of the first temporosphenoidal convolution, and the path of the fibers to the nucleus lies in the posterior part of the internal capsule, the internal geniculate body, the corpora quadrigemina (especially the posterior), and in the lateral lemniscus. (*A*) **Lesions above the nucleus**: (1) Destruction of the center on the left side causes word deafness (words are heard as sounds, with no conception of their meaning). (2) A lesion of the tract between the cortex and the nerve nucleus causes simple deafness. Such lesions may be due to tumor, hemorrhage, softening, etc. (*B*) **Lesion of the nucleus** is rare. (*C*) **Lesion of the nerve** at the base of the brain may be due to meningitis, especially the epidemic cerebrospinal form; to tumors; hemorrhage; trauma; and often is associated with facial paralysis. There may be primary degeneration in locomotor ataxia. (*D*) **Lesion of the labyrinth** may be due to hemorrhage; syphilis; congestion from quinin, or salicylic acid; or inflammation extending from the middle ear or elsewhere, or starting in the labyrinth. (*E*) Hearing may be disturbed by lesions of the middle or external ear, especially wax in the meatus.

Symptoms referable to hearing may be irritative or paralytic: (*A*) **Irritative Symptoms.**—(1) In *hyperacusis* (true auditory hyperesthesia) sounds, even those normally inaudible, are heard with abnormal intensity. This occurs in hysteria, certain cerebral diseases, with stapedius paralysis, meningitis. (2) In *dysacusis* (dysesthesia) ordinary sounds are unpleasant or even painful, as in headache or meningitis. (3) *Tinnitus aurium* denotes various subjective auditory sensations, such as buzzing, clicking, ringing, roaring, etc. It is found in many ear diseases (internal, middle, and external), in neurasthenia, anemia, migraine; as an epileptic aura; from quinin and salicylic acid. (*B*) **Deficient Hearing.**—Usually the fault is not in the nerve, but in other ear structures. If a tuning-fork is heard better when pressed against the temple or mastoid process than when held in front of the ear, the nerve is not at fault.

The Vestibular Nerve.—Disturbances of this branch cause vertigo, nystagmus, nausea, and incoördination of the muscles of the head and eyes.

Meniere's disease (auditory vertigo) is due to disturbance both of the cochlear and vestibular nerves: (*a*) In the internal ear, from hemorrhage or inflammation; (*b*) to a lesion of the nerve itself in tabes or associated with seventh-nerve paralysis; (*c*) to congestion of the labyrinth with disease of the middle ear or of the meatus. **Symptoms.**—Attacks of vertigo and tinnitus, usually with nausea and vomiting, sometimes with momentary unconsciousness. Associated with these attacks is deafness, either partial or complete. The attacks recur at various intervals. Some cases recover; most become completely deaf. The attacks may become more frequent and severe, and the tinnitus or vertigo may become nearly constant. **Treatment.**—Sodium bromid, ʒj per day; quinin or sodium salicylate in very large doses; nitroglycerin; counterirritation over the mastoid. The cause may be amenable to treatment.

QUESTIONS.

Describe the auditory nerve.
Define and give the causes of tinnitus aurium.
Give the causes, symptoms, and treatment of Ménière's disease.

(IX.) GLOSSOPHARYNGEAL NERVE.

This is the motor and sensory nerve to the tonsils, palate, back of tongue, and middle ear. Taste fibers to the posterior third of tongue and palate run with it near the tongue. The motor nuclei may be involved in bulbar paralysis. The nerve or its branches may be involved in meningitis, tumors, trauma, etc. The symptoms of ninth-nerve paralysis are confused by its many communications with other nerves.

(X.) PNEUMOGASTRIC NERVE.

The nucleus is in the medulla, close to the ninth, eleventh, and twelfth nuclei. The nerve trunk receives the bulbar fibers of the spinal accessory nerve. The vagus nerve has a long course and many branches—to the pharyngeal plexus, the larynx, trachea, lungs, heart, esophagus, stomach, intestines, and spleen. It has sensory, motor, and other functions.

Lesions in the **nuclei** are found in bulbar paralysis, usually associated with ninth- and eleventh-nerve lesions. The **nerve trunk** may be involved within the skull in meningitis or tumors or hemorrhage; below the skull, the trunk or its branches may be injured by tumors, aneurysm, operation, or other trauma, or surrounding inflammation. It may be the seat of a neuritis.

Symptoms.—I. **Pharyngeal Branches.**—Motor and sensory nerves to the back of the tongue, the soft palate, and the pharynx. **Motor.**—*Spasm of the pharynx* is usually hysteric. It occurs also in rabies and general convulsions. It causes a sort of gulping and inability to swallow. *Paralysis* occurs in bulbar paralysis, diphtheritic neuritis, or from pressure. If bilateral, swallowing is difficult or impossible, the food regurgitates into the nose or enters the larynx. Unilateral paralysis causes but slight symptoms. **Sensory symptoms.**—*Anesthesia* is common in hysteria.

II. **Laryngeal Branches.**—The superior laryngeal nerve supplies sensation above the vocal cords, and is the motor nerve to the cricothyroid muscle. The recurrent laryngeal nerve is the sensory nerve below the vocal cord and in the trachea, and the motor nerve for all the other laryngeal muscles. **Motor symptoms:** (*A*) *Spasm* of the adductors of the vocal cords constitutes laryngismus stridulus; it occurs in the laryngeal crises of tabes, and in neurotic girls; or it may be induced by attempts at speaking (spastic aphonia). It is a functional disturbance, the symptoms are practically those of laryngismus stridulus (see p. 210). (*B*) *Paralyses of the Larynx.—Lesions:* (*a*) Rarely, there are bilateral lesions in both cortical laryngeal centers. (*b*) There may be a lesion in the nuclei of the nerve, usually bilateral, in bulbar paralysis or tabes. (*c*) A lesion in the nerve trunk or in the recurrent laryngeal nerve is usually unilateral and due to pressure from tumors, large lymph-nodes, aneurysm, or to adhesions from a right apical pleurisy. (*d*) Paralysis is often hysteric, or due to fatigue from long speaking, or associated with laryngitis. (*e*) Rarely there may be a double neuritis as in diphtheria. (1) *Bilateral abductor paralysis:* The laryngoscope shows that in inspiration the cords are not pulled apart, but are forced even nearer together by the current of air, causing a very narrow glottis. The other symptoms are inspiratory stridor and dyspnea, perhaps extreme. Phonation and cough are not affected. (2) *Unilateral abductor paralysis:* One cord is seen near the middle line and does not move in breathing or phonation, the other cord is normal. Voice and cough are hardly affected. (3) *Bilateral adductor paralysis:* The laryngoscope shows that both cords look normal with breathing, but are not adducted in attempted phonation. There is aphonia, but no dyspnea or stridor. (4) *Total bilateral paralysis:* Both cords are moderately abducted (cadaveric position) and motionless. There is aphonia, but no cough, and, on deep inspiration, some stridor. (5) *Unilateral total paralysis:* One cord is moderately abducted (cadaveric position) and motionless. The other cord moves normally, and in phonation is drawn even beyond the middle line to compensate. The other symptoms are hoarseness and a somewhat modified cough.

Sensory Disturbances.—Hyperesthesia is rare. Anesthesia occurs in hysteria, with post-diphtheritic neuritis, or bulbar paralysis.

III. **Cardiac Branches.**—The vagus fibers are sensory, motor, and probably trophic. **Motor function:** (*a*) *Irritation* of the vagi causes slower but strong heart-beats. This may occur with pressure on the nerve in meningitis, tumors, etc. (*b*) *Paralysis* of one vagus often has no effect except a transient vagus stimulation. (*c*) Paralysis of both vagi results in a very rapid heart action, and may occur in neuritis. Emotions may temporarily stimulate or depress the vagi. **Sensory symptoms:** Cardiac palpitation and pain are probably the result of vagus impulses.

IV. The **pulmonary branches** are little understood, but perhaps they are at times responsible for irregularity of breathing.

V. In the **esophagus** spasm and, rarely, paralysis may occur.

VI. **Stomach Branches.**—**Motor.**—*Irritative* disturbance of the motor nerves results in vomiting; usually this is reflex, sometimes it is due to direct irritation of the nerve or its nucleus, as in meningitis. Possibly there may be spasm in gastralgia. *Paralysis* of the stomach is usually of muscular origin. **Sensory disturbances** of the vagus include gastralgia and gastric crises, and may be due to irritation of the nerve ends or of the center. Hunger is supposed to result from vagus impulses.

The **prognosis** of these disturbances depends upon the cause.

Treatment.—The cause is sometimes amenable to treatment. In laryngeal paralyses strychnin and tonics, and in functional cases, electricity are used.

QUESTIONS.

Give the symptoms of pharyngeal spasm and paralysis.

Give the symptoms of laryngeal paralyses.

Give the symptoms from disturbances of the cardiac and gastric branches of the vagus.

(XI.) SPINAL ACCESSORY NERVE.

The **accessory** or bulbar part was described with the vagus-motor functions of the palate and larynx.

The **spinal part** has nuclei in the anterior horn of the cervical cord, the fibers enter the cranium and then come out again. It is the motor nerve to the sternomastoid and part of the trapezius muscles.

Irritative Symptoms.—**Torticollis** (wry-neck) is a disturbance of the sternomastoid and the trapezius chiefly.

(1) *Congenital*, or fixed torticollis, is congenital, but often escapes notice for a few years. It almost always involves the right side. The sternomastoid is short and fibrous, the muscle-fibers are atrophied as a rule. The head is flexed laterally to the right, rotated to the left, and somewhat extended. Associated with this there is facial asymmetry from defective development of the right side and spasm of the sound side. Sometimes the trapezius also is involved. Tenotomy cures the wry-neck, but the facial asymmetry persists, and may even seem aggravated.

(2) *Spasmodic Wry-neck.*—Usually in males and in middle life; it may follow trauma, exposure to cold or no discoverable cause; is more common in those of a neurotic tendency; it may occur in hysteric women; with caries of the cervical spine, or large tender nodes in the neck. It is sometimes a

pure neurosis. (*A*) There is spasm in the sternomastoid, and the head is bent laterally, extended dorsally and the face rotated to the other side. If the trapezius also is involved the occiput seems pulled down and the shoulder raised. The contracted muscles are hard, tense, often prominent and hypertrophied. The spine may be flexed laterally, concave toward the affected side. The spasm may be tonic and last for weeks. (*B*) It may be a series of single contractions, recurring every few minutes (called clonic wry-neck), and here the splenius and other deep neck-muscles are often involved. The spasm is absent in sleep. (*C*) Bilateral spasm may occur—if tonic the head is extended; if intermittent and synchronous on both sides the head is jerked backward, and often the shoulders are raised; if the two sides contract at different times there is a complex rotary shaking of the head. Tonic and clonic spasm may alternate in the same person.

Prognosis.—Tonic spasm usually disappears in time. Clonic spasm is apt to persist, with remissions, throughout life, being worse at times of fatigue or intense emotion. The spasm may spread to the face and arms.

Treatment.—Bromids may relieve for a time. Morphin often helps, but there is great danger of habit. The ascending galvanic current is used. Neurectomy of the spinal accessory or the posterior cervical nerve roots may help.

Paralysis of the Spinal Part.—**Lesions**: (*a*) of the nucleus occur in progressive muscular atrophy and are bilateral; (*b*) of the nerve roots with meningitis, with caries of the cervical spine or tumors; (*c*) of the trunk in cervical caries, abscesses, trauma, etc. There is paralysis of the sternomastoid, causing imperfect rotation of the head toward the other side, but no torticollis. Also of that part of the trapezius which runs from the occiput to the acromion; this causes drooping of the shoulder and, because the shoulder is not properly fixed, defective raising of the arm. If bilateral paralysis of the sternomastoid exists the head falls backward. With paralysis of both trapezii it falls forward. The paralyzed muscles atrophy and show R. D. The cause of pressure may be amenable to treatment. Electricity and massage may help the muscles.

QUESTIONS.

Give the etiology, varieties, symptoms, and treatment of wry-neck.

Give the symptoms of paralysis of the spinal part of the eleventh nerve.

(XII.) HYPOGLOSSAL NERVE.

The cortical center is probably in the lower part of the anterior central gyrus; the nucleus in the floor of the fourth ventricle in the medulla. It is the motor nerve of the tongue and most of the muscles of the hyoid bone.

Irritative Symptoms.—**Spasm** occurs as part of a general convulsion or in chorea or facial spasm. It may occur in hysteria, with stuttering, or reflexly from irritation of the fifth nerve. It may be unilateral or bilateral, tonic or clonic.

Paralysis.—(1) A lesion above the nucleus is common as part of hemiplegia. There is no wasting or change of electric reaction. (2) The nuclei may be damaged by hemorrhage, or in tabes or bulbar paralyses, usually bilaterally. (3) The nerve may be injured in meningitis, with hemorrhage, by tumors, with disease of bone at the foramen, or lower down by scars, tumors, trauma, etc. **Symptoms.**—*Unilateral Paralysis:* The tongue, when protruded is pushed *toward* the affected side. The affected half is flabby, atrophied, wrinkled, gives R. D. Swallowing, chewing, speaking are not much changed. Taste and sensation are unaffected. *Bilateral Paralysis:* The tongue lies in the floor of the mouth motionless. It can not be moved voluntarily, is wrinkled, flabby, shows R. D., and perhaps fibrillary twitchings. Swallowing, chewing, and lingual sounds (*t, d, th, l, r, s, j, ch,* etc.) are much impaired. The prognosis of paralysis is bad.

QUESTIONS.

Give the causes of spasm of the tongue.

Give the causes and symptoms of paralysis of the tongue.

SPINAL NERVES.

PHRENIC NERVES.

The nucleus is in the anterior horn of the fourth, and either the third or fifth cervical segments. It is the motor nerve of the diaphragm.

Hiccough.—This is a sudden short spasm of the diaphragm, associated with sudden closure of the glottis. **Causes:** (1) Inflammation of abdominal structures—peritonitis, intestinal obstruction, gastritis, appendicitis, typhoid, etc. (2) Irritation of the diaphragm from gastric distention. (3) Neurotic—in hysteria, epilepsy, cerebral tumors, shock, etc. (4) It occurs also in uremia, gout, perhaps after violent laughter and often spontaneously. Sometimes it persists for days or weeks and exhausts the patient.

Paralysis.—The lesion may be (*a*) in the nucleus and is usually bilateral, or (*b*) in the nerve, usually unilateral, from pressure by tumors, or aneurysm; from wounds; occasionally neuritis, in diphtheria, lead-poisoning, etc. The paralysis may be partial or complete. If **complete,** the patient may be comfortable when still; the breathing is purely costal; activity induces marked dyspnea; the abdominal movements are reversed, retracting in inspiration. There is marked tendency to bronchitis, edema, and pneumonia at the bases. Coughing is impeded. **Unilateral paralysis** is hard to make out, but the fluoroscope will show it. Motion of the diaphragm may be stopped without paralysis with pleurisy either diaphragmatic or with large effusions. *Prognosis:* Phrenic paralysis is usually a part of a serious disease, such as anterior poliomyelitis or multiple neuritis, and many cases die.

QUESTION.

Give the symptoms of bilateral paralysis of the diaphragm.

BRACHIAL PLEXUS.

The nerves come from the fourth cervical to the second thoracic segments of the cord. Disease above the nuclei is not considered here. With disease of the lower motor-segment there are the trophic and electric changes, prognosis, etc., described under Neuritis (see p. 328).

Paralysis of Special Nerves.—Long Thoracic Nerve.—Serratus magnus palsy usually occurs in men, before middle life, on the right side; (*a*) from pressure in the posterior triangle of the neck (hod-carriers, etc.); (*b*) also in plasterers;

(*c*) it may be involved in neuritis from exposure; (*d*) in progressive muscular atrophy; (*e*) anterior poliomyelitis. The affected scapula is "winged," its inner border and the angle project; this is exaggerated in pushing with the arm. Neuralgic pains precede or accompany the onset. The course is usually protracted over many months.

The **circumflex nerve** may be injured by pressure of a crutch, with dislocation of the humerus, or a fall on the shoulder. This leads to paralysis of the deltoid and teres minor. The arm can not be raised, the deltoid looks very flat, there may be anesthesia over this muscle and trophic changes in the shoulder joint, with thickening of the ligaments and restriction of movement.

Musculospiral paralysis is very common and may be due to blows; fractures; pressure upon the nerve during sleep or anesthesia; to neuritis from cold; lead and other poisons. The nerve supplies the triceps, brachialis anticus, the supinator longus and brevis, and the extensors of the wrist and fingers: (*a*) Rarely all of these muscles are paralyzed, but usually the lesion is too low to involve the brachialis anticus and triceps. (*b*) The paralysis from pressure during sleep usually involves the supinators and extensors and is unilateral. (*c*) The toxic neuritis is bilateral, and the supinator longus and extensor carpi radialis longior escape (at least in lead-poisoning). All have wrist-drop (see p. 331). The sensory symptoms are slight. These palsies may last only a few days or for months.

The **ulnar nerves** may be damaged by pressure, stretching or trauma near the elbow and in fractures or dislocations, or in neuritis. In flexion of the wrist there is radial abduction of the hand (paralysis of flexor carpi ulnaris); there is loss of flexion of the distal joints of the fourth and fifth fingers (flexor profundus digitorum, ulnar half), but not of the other finger joints; loss of adduction of the thumb (adductor pollicis); loss of flexion of the metacarpophalangeal joints, of extension of the interphalangeal joints (especially of the fourth and fifth fingers), and of adduction and abduction of the fingers (interossei and third and fourth lumbricals and the muscles of hypothenar eminence, which are wasted). In time a moderate degree of claw-hand develops. *Sensation* is lost on the ulnar side of the hand, the back of two and one-half fingers and the front of one and one-half fingers.

The **median nerve** may be the seat of injury or neuritis. It supplies the flexor sublimis and radial half of the flexor profundus digitorum, the abductor and flexors of the thumb, the two radial lumbricals, pronators, and flexor carpi radialis. With *paralysis*, pronation stops at the midposition; the wrist in flexion is pulled to the ulnar side; the thumb can not be opposed to the fingers; there is loss of flexion of all the second phalanges and of the distal phalanges of the fourth and middle fingers. The thenar eminence is atrophied. There is *loss of sensation* over the radial side of the palm, of the palmar surface of the thumb, and of the fingers not supplied by the ulnar.

Combined Paralysis of the Brachial Plexus.—Many combinations of paralysis of several nerves of this plexus occur. The more important forms are: (1) Due to dislocation of the humerus, especially subcoracoid, or a blow on the shoulder. This may cause a slight and transient, or a permanent, paralysis of some or all of the branches of the plexus. (2) Erb's supraclavicular point is a point corresponding to the fifth and sixth cervical nerve roots and is found over the sixth cervical transverse process, just behind the external border of the sternomastoid, a little above the clavicle. Electric stimulation here causes contraction of the deltoid, biceps, supinator longus, and sometimes of the supraspinatus, infraspinatus, and supinator brevis. **Erb's or Duchenne's paralysis** involves these muscles and may be due to injury at birth. (3) **Brachial neuritis** may be primary in the aged and gouty. The symptoms are intense pain, paroxysmal or continuous, over the plexus and its distribution, increased by moving the arm. The nerves are tender.

QUESTION.

Give the causes and symptoms of paralysis of the various nerves of the brachial plexus.

LUMBAR PLEXUS.

The **obturator nerve** to the adductors of the thigh may be injured during parturition. The affected thigh can not be crossed over the other, or rotated outward. The muscles atrophy.

The **anterior crural nerve** may be injured in wounds, parturition, or dislocation of the hip. The quadriceps extensor is paralyzed and atrophied; the knee can not be extended; there is anesthesia of the front and inner side of the thigh and the inner side of the leg and foot. Pain may be an early symptom.

Gluteal nerve paralysis affects the gluteus medius and the minimus with loss of abduction of the thigh.

Combined paralysis of the lumbar plexus may be due to psoas abscess, diseased lymph-nodes or disease of bone.

QUESTION.

Discuss paralyses of the nerves of the lumbar plexus.

SACRAL PLEXUS.

The **external popliteal nerve** may be injured by trauma or neuritis. It supplies the peronei, long and short extensors of the toes and the tibialis anticus, and is the sensory nerve for the outer half of the front of the leg and dorsum of the foot. Paralysis of the nerve causes anesthesia here and foot-drop with steppage gait. Subsequent contractures of the calf muscles may lead to pes equinus.

Lesions of the **internal popliteal nerve** lead to paralysis of the popliteus, of the calf, of the long flexor of the toes, and the muscles of the sole; with loss of plantar flexion of the ankle and of flexion of the toes. Talipes calcaneus and claw foot may develop from contractures.

Great Sciatic Nerve.—This is the motor nerve for the flexors of the knee and all the muscles below the knee, and the sensory nerve for the outer half of the leg and foot.

Irritative Lesions.—**Sciatica.**—Usually in adults and in males. It occurs in the gouty; the rheumatic; after exposure to cold and wet; from pressure within the pelvis from ovarian, uterine or other tumors, or from the fetal head in labor; or from pressure at the great sciatic notch, or in the thigh; from inflammation of the hip, etc.

Lesion.—The nerves that have been seen showed perineuritis or interstitial neuritis (see p. 328), most marked at the notch or middle of the thigh.

Symptoms.—Pain is the most prominent symptom; it is referred to the course of the nerve; is boring, often intense; may be worse at night; is increased by stretching the nerve, for instance, in flexing the thigh and when the knee is extended or by walking. In severe cases the patient walks on tiptoe with the knee bent. There is tenderness over the whole nerve, most marked between the great trochanter and the ischium, at the middle of the thigh, in the popliteal space, below the head of the fibula, and behind the external malleolus. The spontaneous pain also is greatest at these points.

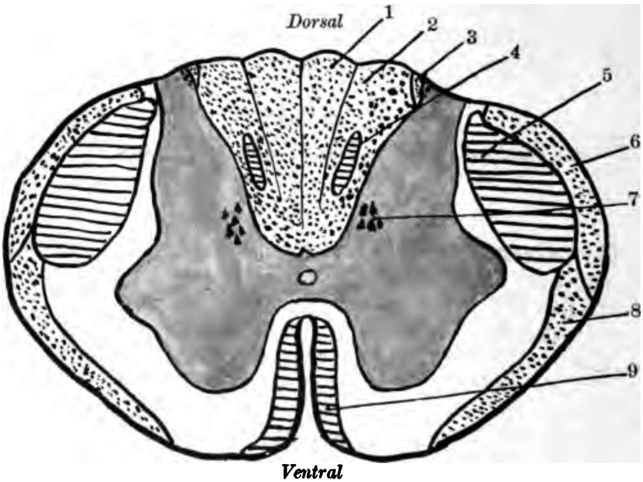

FIG. 2.—Diagram of the spinal cord. The tracts that degenerate upward (ascending degeneration) are stippled, those that degenerate downward (descending degeneration) are shaded with lines: 1, Posterior median column (Goll); 2, posterior lateral column (Burdach); 3, column of Lissauer; 4, comma bundle; 5, crossed pyramidal column; 6, direct cerebellar tract (Flechsig); 7, Clarke's column (gray matter); 8, anterolateral ascending tract (Gowers); 9, direct pyramidal column (Türck).

Course.—Sometimes there is fever at the start. The pain usually begins above and spreads downward; it may last weeks or months, and in the protracted cases there is atrophy of the muscles, probably from disuse, the reaction of degeneration is very rare; anesthesia is rare. Often the patient is bedridden for weeks. Sciatica is usually unilateral, but rarely is bilateral. After recovery it is apt to recur. The neuritis may ascend to the sacral plexus or to the spinal cord, but this is rare.

Treatment.—The cause should be carefully sought in the pelvis, hip joint, spine, and in the general diathesis of the patient, and should be treated. The patient should rest in bed. Fixation of the limb by a long splint or extension apparatus is often used; also hydrotherapy, especially at hot springs. Locally, over the nerve, especially at the tender points, use blisters, the cautery, the ascending galvanic current, deep injections into the nerve of cocain or morphin or pure water; acupuncture often helps, the needle being thrust into the nerve and left there fifteen to thirty minutes. The weakened muscles are helped by electricity and massage. After the acute stage the nerve may be stretched.

Paralysis of the great sciatic nerve follows severe injury to the nerve in the spinal canal from tumors, etc. (usually bilateral); or in the pelvis or along the course of the nerve (usually unilateral). There is paralysis of motion and sensation. If one nerve only is affected the quadriceps extensor keeps the knee stiff and extended, and walking is possible.

Combined paralysis of the sacral plexus may follow injuries; tumors and inflammation in the pelvis; or parturition. Neuritis may extend upward to the plexus, usually from the great sciatic nerve.

QUESTIONS.

Give the causes, lesions, symptoms, prognosis, and treatment of sciatica.

Discuss the other disturbances of the nerves of the sacral plexus.

SPINAL CORD.

The motor and sensory tracts and their trophic relations are described on page 321. The localization of various functions and their reflexes are given in the following table:

Localization of the Functions of the Segments of the Spinal Cord.

SEGMENT.	MUSCLES.	REFLEXES. (Manipulation=Effect)	SENSATION.
II. & III. Cervical.	Sternomastoid, trapezius, scaleni and neck, diaphragm.		Back of head to vertex; neck.
IV. Cervical.	Diaphragm, deltoid biceps, coracobrachialis, supinator longus, rhomboid, supra- and infraspinatus.	Pupil—IV. to VII. Cervical (or II. Thoracic): Irritation of neck = dilation of pupil.	Neck; upper shoulder; outer arm.
V. Cervical.	Deltoid, biceps, coracobrachialis, brachialis anticus, supinator longus and brevis, rhomboid, teres minor, pectoralis (clavicle part), serratus magnus.	Scapular—V. Cervical to I. Thoracic: Irritation of skin over scapula=contraction of scapular muscles. Supinator longus: Tapping its tendon=flexion of forearm.	Back of shoulder and arm; outer side of arm and forearm, front and back.
VI. Cervical.	Biceps, brachialis anticus, pectoralis (clavicle part), serratus magnus, triceps, wrist and finger extensors, pronators.	Triceps—V. to VI. Cervical: Tapping elbow tendon = extension of forearm. Posterior wrist, VI. to VII. Cervical: Tapping extensor tendons=extension of hand.	Outer side of forearm, front and back; outer half of hand.
VII. Cervical.	Triceps (long head), wrist and finger extensors, wrist pronators, wrist flexors, subscapular, pectoralis (costal part), latissimus dorsi, teres major.	Anterior wrist—VII. to VIII. Cervical: Tapping anterior tendons = flexion of wrist. Palmar—VII. Cervical to I. Thoracic: Stroking palm =closure of fingers.	Inner side and back of arm and forearm; radial half of hand.
VIII. Cervical.	Flexors of wrist and fingers, muscles in the hand.		Forearm and hand, inner half.
I. Thoracic.	Extensors of thumb, muscles in hand.		Forearm, inner half; ulnar nerve to hand.
II. to XII. Thoracic.	Muscles of back and abdomen, erectores spinæ.	Epigastric—IV. to VII. Thoracic: Tickling mammary region= retraction of epigastrium. Abdominal—VII. to XI. Cervical; Stroking flank= abdominal retraction. Vasomotor centers—II. Thoracic to II. Lumbar.	Skin of chest and abdomen in zones sloping downward; navel (X. Thoracic); upper gluteal region.

Segment.	**Muscles.**	**Reflexes.** (Manipulation=Effect)	**Sensation.**
I. Lumbar.	Iliopsoas, sartorius, muscles of abdomen.	Cremasteric—I. to III. Lumbar. Stroking inner thigh=retraction of testis.	Skin over groin and front of scrotum.
II. Lumbar.	Iliopsoas, sartorius, flexors of knee, quadriceps femoris.	Patellar tendon (knee-jerk)—II. to IV. Lumbar: Tapping tendon=a kick.	Outer side of thigh.
III. Lumbar.	Quadriceps femoris, inner rotators of thigh, abductors of thigh.		Front and inner side of thigh.
IV. Lumbar.	Abductors of thigh, adductors of thigh, flexors of knee, tibialis anticus.	Gluteal—IV. to V. Lumbar: Stroking buttock = dimpling in fold of buttock.	Inner side of thigh, leg and foot.
V. Lumbar.	External rotators of thigh, flexors of knee, flexors of ankle, extensors of toes.	(Some say Achilles tendon.)	Back of thigh and leg and outer part of foot.
I. to II. Sacral.	Calf, flexors of ankle, long flexor of toes, peronei, muscles in foot.	Plantar: Tickling sole =flexion of toes and leg retraction. Baginski (extensor plantar reflex): Tickling sole=extension of great toe.	Back of thigh, leg and foot, outer side.
II. to V. Sacral.	Perineal muscles. Muscles of bladder and rectum.	Foot reflex (Achilles tendon): Forcible dorsal flexion of foot =ankle clonus. Bladder and rectal centers.	Skin over sacrum, anus, perineum, external genitals.

Sensory Centers of the Viscera (Head).

Heart—II. to VIII. Thoracic.
Lungs—III. and IV. Cervical, III. to IX. Thoracic.
Stomach—VI. to X. Thoracic (Cardiac end, VI. and VII. Thoracic; pyloric end, X. Thoracic).
Intestines, except rectum—X. to XII. Thoracic.
Rectum—II. to IV. Sacral.
Liver and gall bladder—VII. to X. Thoracic.
Kidney and ureter—X. Thoracic and I. Lumbar.
Bladder muscle—XI. Thoracic, I. Lumbar.
Bladder mucosa and neck—II. to IV. Sacral.
Testis or ovary—X. Thoracic.
Uterus—X. Thoracic, I. Lumbar.
Os uteri—II.—IV. Sacral.

SECONDARY DEGENERATIONS (See also p. 323).

Secondary descending degeneration in the spinal cord affects the **motor tract** chiefly. With a lesion in the superior segment in the cortex the entire neuron degenerates, including the whole length of its pyramidal fiber. A lesion at any point of the fiber leads to degeneration of that fiber below the lesion. Degeneration of the lower motor segment has been discussed (see p. 329), and is precisely similar. Descending degeneration of **sensory fibers** is found in the comma tract. A lesion of the posterior spinal ganglion, or the posterior nerve root central to the ganglion, or the descending branches of these axons in the cord leads to descending degeneration of the terminal part of the axon which is short.

Ascending Degeneration.—(1) **Column of Goll**: Most of these fibers come from cells of the posterior root-ganglion and end in the nucleus gracilis. They degenerate upward to the nucleus gracilis after a lesion of the posterior root-ganglion, of the nerve root central to it, or a lesion of the cord involving this column. (2) **Column of Burdach**: The cells are in the posterior root-ganglion; many fibers run to the nucleus cuneatus and the olive. Degeneration is like that of the fibers of the column of Goll. (3) **Lissauer's column**: The cells are in the posterior root-ganglion, the fibers run upward, and degenerate like the previous columns. The fibers of the columns of Goll, Burdach, and Lissauer do not cross the middle line. (4) **Direct cerebellar tract**: The cells are in the column of Clarke, in the posterior horn, on the *other* side of the cord. The axons soon cross the middle line, run to and through the restiform body to the superior worm of the cerebellum. This column is not found below the thoracic cord. Degeneration occurs with a lesion of Clarke's column on the other side of the cord or a lesion in the fibers. (5) **Antero-lateral ascending tract of Gowers** is not perfectly understood. Cells are probably in the posterior root-ganglion of the other side, the fibers soon cross the middle line, run to the cerebellum, through the medulla, pons and superior peduncle. It degenerates after a lesion of the ganglion on the other side or above a lesion in the axon. The exact paths of the ordinary sensory impulses are by no means perfectly known, but anesthesia of one side of the body is associated with a lesion in the other side of the cord.

Classification of Diseases.—Those neurons having similar functions constitute a **system**; thus the *efferent system* carries efferent impulses, and comprises all the motor tract from the cortical cell to the end plates. The *afferent system* is more complex; it includes the sensory portions of the peripheral nerves, the posterior roots and ganglia, the neurons and axons concerned in ascending degeneration, and very many parts of the brain. Certain diseases cause *primary degeneration*, wholly or chiefly in the neurons of one of these systems, and are called **system diseases**—*e. g.*, anterior poliomyelitis and primary lateral sclerosis are limited to the efferent system, and locomotor ataxia is limited to the afferent system. If a disease attacks neurons of both systems it is called a **combined disease**—*e. g.*, ataxic paraplegia. A **focal disease** attacks a single, rather small, circumscribed area of the cord or brain, but, of course, secondary degeneration may be extensive. **Insular** or **disseminated diseases** attack several separated foci. A **diffuse disease** involves a single area of considerable size. A **transverse lesion** involves the whole thickness of the cord at one level, like a cross-section.

Total transverse lesion of the cord destroys certain neurons and also cuts off all nervous connection with the brain from parts below the lesion. This would cause (*A*) **Sensory symptoms**: (1) Complete anesthesia of the body below the lesion, but no trophic symptoms. There might be (2) irritative symptoms (girdle sensation) in the distribution of the nerve rising just above the lesion, and (3) anesthesia and trophic disturbances corresponding to the cells or root-fibers destroyed at the lesion. Therefore, if the sensory supply of the body is known, the line between anesthesia and hyperesthesia will locate the site of the lesion. (*B*) **Motor symptoms**: If the destroyed cells happen to innervate muscles easily studied, these would show changes due to a lesion of the lower motor segment (see p. 326). The muscles connected with the lower parts of the cord would show the changes due to a lesion in the upper segment only (except that in complete transverse lesion of the cord the reflexes may be abolished below the lesion). If the lesion is extensive the symptoms from the destruction of the cells and nerve roots (muscular atrophy, R. D., trophic disturbances of skin, etc.) would be marked enough to allow of the determination of the lower, as well as of the upper limit of the lesion.

Unilateral Transverse Lesion (Brown-Séquard Paralysis).—*On the Side of the Lesion.*—**Sensory symptoms**: Just above the lesion cutaneous hyperesthesia. Corresponding to the injured segment a zone of anesthesia. Below this there may be loss of muscular sense, but hyperesthesia of tactile and temperature sense. **Motor Symptoms.**—Below the lesion: upper segment paralysis; no trophic disturbances; deep reflexes increased, at least after a few days. On *the other side of the body* the only change is cutaneous anesthesia below the level of the lesion.

Sensory paralysis may not include all sensations, but only one or more kinds (elective sensory paralysis). There is often loss of pain and temperature sense, while touch persists, or there may be loss of muscular sense alone, or many other combinations. It frequently happens that sensory symptoms are hard to understand or explain.

QUESTIONS.

Give the secondary degenerations of the cord, ascending and descending.

What is a system disease? Give examples.

Give the symptoms of a total transverse lesion of the cord. How are the site and the limits of the lesion determined?

Give the symptoms of Brown-Séquard paralysis.

SPINAL MENINGITIS.

External meningitis involves the outer surface of the spinal dura. **Internal meningitis** begins inside of the dura. **Pachymeningitis** involves the dura. **Leptomeningitis** involves the pia.

ACUTE SPINAL MENINGITIS.

Etiology.—(1) A part of cerebrospinal meningitis, epidemic or tubercular; (2) rarely it is found in pneumonia, typhoid or other infection; (3) it may follow empyema; (4) possibly it may be primary after exposure. It is a bacterial infection.

Lesions.—An exudative, often purulent, inflammation of the spinal pia (see p. 55).

Symptoms.—This often is merely a part of meningitis, as described on p. 55. The symptoms suggestive of spinal involvement are: (1) **Irritative Symptoms.**—*Sensory:* Intense pain along the spine, continuous, aggravated by movement, pressure or local heat; paroxysms of pain along the nerve trunks and their distribution; hyperesthesia. *Motor:* Convulsions are usually cortical, but spinal or nerve-root irritation may be responsible for tonic spasm of the back muscles and the rigid spine, the retracted belly, sometimes spasm of other muscles, and also Kernig's sign. *Vasomotor:* The tache cerebrale. *Reflexes* may be increased. (2) **Paralytic symptoms** may appear later, such as anesthesia, lessened reflexes, and especially constipation and retention of urine, which are often present from the onset. The back muscles seldom show relaxation, but incontinence of urine and feces often appears. If the meningitis involves the cervical cord, the vagus, phrenic and sympathetic disturbances may affect the respiration, the pupil, and the heart. Protracted or permanent paralysis of muscles (lower-segment type) may follow extensive involvement of nerve roots. Lumbar puncture (p. 58) is an important aid to diagnosis.

Prognosis.—Except in cases due to the meningococcus, recovery is improbable.

CHRONIC LEPTOMENINGITIS.

This may follow chronic alcoholism, syphilis, trauma, or chronic diseases of the cord. The pia is opaque, thickened, and adherent to the cord and dura. Thick gelatinous matter fills the arachnoid spaces. There are usually no noticeable symptoms, except those of the primary disease of the cord.

CHRONIC INTERNAL PACHYMENINGITIS.

(1) **Hypertrophic cervical pachymeningitis** is rare. The dura is thick laminated connective tissue, perhaps one-fourth inch thick. This compresses the motor roots of the lower cervical nerves, and leads to atrophy of the ulnar and median nerves; later, pressure on the cord cuts off the

pyramidal tracts. (1) **Painful period**: Neuralgic pains in the arms for two or three months. (2) **Paralytic period**: Lower-segment paralysis of the muscles supplied by the ulnar and median nerves (p. 351). Contraction of unopposed muscles causes a characteristic position of the hand; the wrist is overextended, and the fingers partly flexed. Anesthesia may be present. (3) **Spastic paraplegia** (upper-segment paralysis) follows when the cord communications are cut off. Anesthesia and trophic disturbances (bedsores, etc.) may develop. Death follows from exhaustion.

(2) **Internal hemorrhagic pachymeningitis** is found in the insane and alcoholics, associated with the same condition in the cranium.

HEMORRHAGE INTO THE SPINAL MENINGES.

Hemorrhage may be extrameningeal, outside the dura; or intrameningeal, within the dura; and this latter may be subdural or subarachnoid. It may be due to trauma, hemorrhagic diseases, convulsions, rupture of an aneurysm, erosion of a vessel in caries, etc. Minute hemorrhages may occur with infectious diseases.

Symptoms.—If the hemorrhage is large and sudden enough, it causes severe pain in the back and paroxysmal pain along nerves whose roots are involved. Soon the symptoms of a transverse lesion of the cord, unilateral or complete (p. 359), appear. There is no fever or head symptoms. It usually kills in a few hours.

Treatment.—There is the general treatment for hemorrhage; the patient should lie prone; an operation is indicated.

QUESTIONS.

Give the etiology and symptoms of acute internal spinal meningitis. Discuss chronic spinal meningitis.

Give the causes and detail the symptoms of hemorrhage into the spinal meninges.

HEMORRHAGE INTO THE SPINAL CORD (Hematomyelia).

This is rare. It may be due to trauma, arterial disease, meningitis, or convulsions.

Small hemorrhages may occur and form pigmented scars.

Large hemorrhage may be unilateral, involving the gray matter only; or more extensive, involving the white matter also; or it may be bilateral. There are the symptoms of partial or complete transverse lesion of the cord if the clot is large enough. Usually paralysis comes on at once. Many cases recover. Small hemorrhages are not recognized clinically.

QUESTION.

Give the causes and symptoms of hematomyelia.

Congestion and *anemia* of the spinal cord are not recognized clinically.

SLOW COMPRESSION OF THE CORD.

Causes.—(1) Caries of the spine (Pott's disease) is the chief cause. This is more common in children, may follow trauma, and is tuberculous. (2) Carcinoma and sarcoma of the vertebræ, usually secondary to scirrhus of the breast or stomach; chronic cervical meningitis; aneurysm of the thoracic or abdominal aorta that erodes the spine; echinococcus in the spinal canal; tumors of the cord or its membrane, are occasional causes.

The **lesion** results in gradual compression of the cord. This may be from a knuckle of bone in angular deformity of the spine; thickened periosteum or dura; caseous masses; or the various new growths. Ordinary kyphoscoliosis without caries does not cause pressure. At the affected area, usually only an inch or two long, the cord is thinner, usually softer. Microscopically it often shows surprisingly little change, but in time there is degeneration, chiefly in the white matter, with increase of neuroglia. In very old cases the cord at the compressed point may be slender, hard, and composed of dense fibrous tissue. There are also the secondary degenerations above and below. With tumors, nerve roots may be involved in the compression. Recovery may occur even after a long time.

Symptoms.—The primary condition must be sought. Probably it has existed for a long time. In Pott's disease there are angular deformity, tenderness upon pressure in the axis of the spine and upon direct pressure over the lesion, muscular spasm of the back muscles when the spine is extended, and other symptoms. Usually the lesion is in the cervical or upper thoracic regions, sometimes lower down.

Nerve-root Symptoms.—At the level of the lesion two or three roots on one or both sides may be irritated, causing severe and lancinating pain along the course and distribution of the nerve. This is most common and severe in cancer. There may be intense pain in regions that are anesthetic (anesthesia dolorosa). *Trophic symptoms*, especially herpes, may be present. *Motor symptoms:* At first the reflexes may be increased, later there is weakness or a lower-segment paralysis if the cervical or lumbar spine is involved. In dorsolumbar Pott's disease there may be retention, and later incontinence, of urine and feces; also bedsores.

Cord Symptoms.—The symptoms of a transverse lesion of the cord (see p. 359) may be more or less marked. With a lesion below the second lumbar vertebra, no cord symptoms are present, merely nerve-root symptoms. The cord transmits impulses to and from the brain for a surprisingly long time, and the sensory impulses are more persistent than the motor. *Motor symptoms:* Reflexes often are exaggerated. Sometimes there is distinct upper-segment paralysis. The bladder and rectum are occasionally affected. In long-standing and very severe cases the transverse lesion may be complete.

Course.—Usually irritative sensory symptoms from involved nerve roots appear first; then irritative motor symptoms at this level. After a considerable time symptoms below this level may appear, perhaps associated with paralytic symptoms at the level of the lesion. Recovery may occur after even a year of spastic paralysis, and is explained by the persistence of the fibers noted above. Many cases die of tuberculosis, cancer, bedsores, cystitis, pyelonephritis or exhaustion.

Treatment.—There is the special treatment of the cause; also protracted rest in bed. Operation seems to have encouraging results in certain cases. Besides this the treatment is symptomatic.

CAUDA EQUINA AND CONUS MEDULLARIS.

Below the second lumbar vertebra injuries and tumors are common, but they do not, of course, involve the spinal cord itself. They sometimes cause some paralysis of bladder and rectum alone or with a slight patch of anesthesia around the coccyx, perineum, and external genitals; more commonly motor and sensory paralysis involving various lumbar and sacral nerves on one or both sides is seen.

NEW GROWTHS OF THE CORD AND MEMBRANES.

(1) Extradurally there may be lipoma or parasites. (2) Within the dura the membranes may be the seat of fibroma, sarcoma, gumma, tuberculous growth, etc. (3) In the cord or on its surface there may be tuberculous or syphilitic growths, glioma, etc. The tumor slowly compresses the cord, with softening and secondary degeneration. It may cause syringomyelia or myelitis. The nerve roots are often involved.

Symptoms.—Symptoms are often absent or there may be the symptoms of slow compression of the cord. With a tumor of the meninges the nerve-root symptoms usually appear first. With a tumor of the cord itself the first symptoms usually are those of destruction of the gray matter or of the white matter; then of a unilateral transverse lesion; and finally of a complete transverse lesion. Myelitis often complicates. The site of the tumor may be diagnosed in the usual way. Its nature may sometimes be inferred from the study of the etiology, and the presence of tuberculosis, syphilis or cancer elsewhere.

Treatment.—The cause may perhaps be amenable to treatment. Excision of the tumor may be possible. Symptomatic treatment is indicated.

QUESTIONS.

Give the causes, lesions, symptoms, and treatment of slow compression of the cord.

Discuss lesions of the cauda equina and conus medullaris.

What tumors involve the cord and its membranes?

What symptoms do they cause?

SYRINGOMYELIA.

Etiology.—Is more common in males, usually beginning before thirty. **Lesions**: This is a **gliosis** with the formation of embryonic neuroglia tissue, and hemorrhage or degeneration resulting in a **cavity.** It may involve a small part of the cord (usually cervical or thoracic), or more, even all the cord. At different levels it may involve different parts of the cord, at one level the posterior horn, somewhere else other parts of the gray or white matter. The diameter of the cavity may be almost microscopic, or, reversely, as much as half an inch. The whole cord looks larger, and it may fluctuate. On incision, clear flocculent or bloody liquid may escape and the cord collapses. The wall of the cavity is dense neuroglia. The vessels are thickened, often thrombosed, pigment is common. There may be destruction of the gray or white matter of the cord with secondary degenerations.

Symptoms.—There may be no symptoms even with a big cavity. (1) The *most typical cases* often begin in the cervicothoracic region, with pain; later, pain and temperature sense are lost, with a consequent tendency to burns and other injuries, while touch persists; there is *lower-segment* muscular paralysis, beginning in the hands and extending to the arms. **Other symptoms**: There may be spastic paralysis of lower levels, and later this may change to the lower-segment type. Kyphoscoliosis is common. Trophic and vasomotor symptoms are not rare, such as bullæ, perforating ulcer, spontaneous fractures, swollen painless joints. The medulla also may be involved with paralysis of its nerves, and perhaps glycosuria and polyuria.

Certain cases have motor symptoms only for years. In other cases sensory symptoms may predominate, with pain and temperature anesthesia. Morvan's disease is a type with neuralgic pain, loss of all sensation, painless whitlows (felons), ulcers, and muscular atrophy.

The **course** is very chronic: patients die of an intercurrent disease, emaciation, bulbar paralysis, bedsores or infections.

The **treatment** is symptomatic.

Other Cavities of the Cord.—Hydromyelia is a dilated central canal lined, at least in part, with columnar epithelium. Myelitic foci or tumors may soften and form cavities.

QUESTION.

Give the lesions and symptoms of syringomyelia.

MYELITIS.

ACUTE MYELITIS.

Etiology.—Myelitis is more common in males and in young adults. It is probably an infection. It may follow exposure, especially when drunk; the puerperium; measles; variola; trauma or disease of the vertebræ; tumors or syphilis of the cord; or an ascending neuritis.

Lesion.—The lesion may be **focal, disseminated** or **diffuse** (p. 359). It may attack the gray matter only, or the white only. If it involves the whole section of the cord it is *transverse myelitis.* The involved part of the cord shows a reddened pia. The cord is swollen, soft, sometimes diffluent on section; congested or hemorrhagic; may be reddish or brown, yellow or gray. Microscopically there are the products of exudation; degeneration of the nerve fibers and cells; and later proliferation of the neuroglia and secondary degeneration upward and downward. The most common site is the lumbar and thoracic region.

Symptoms.—(1) **Acute transverse myelitis** is the most common form. At the *onset* there are fever and tingling or moderate pain in the legs. Soon there is weakness of the legs, developing into complete paralysis within a few hours or days. (*a*) The usual site is the *thoracic cord,* and there are the symptoms of a complete transverse lesion here (p. 359); Babinski's sign is present. *The bladder:* The detrusor is first weakened (difficult micturition); later, it is paralyzed (retention); still later the sphincter is paralyzed with the incontinence of constant overflow. This often leads to cystitis and pyelonephritis. *The rectum:* Constipation at first, later incontinence of feces. Bedsores are common, partly from maceration of the skin in urine and feces, and sometimes from trophic disturbances. (*b*) *Cervical transverse myelitis* gives lower-segment paralysis of muscles of the arms, later anesthesia also; spastic paralysis of the lower limbs; bladder and rectal disturbances. Also there may be vomiting, hiccough, slow pulse, paralysis of the phrenic nerve, myosis. (*c*) *Lumbar myelitis* causes lower-segment paralysis of the muscles of the lower limbs; bladder and rectum disturbances; later anesthesia and very marked trophic symptoms, especially bedsores. *Course of transverse myelitis:* Some recover soon or late. The disease may stop but leave permanent spastic paraplegia. Many die of infections of the lungs, kidneys and bladder, or bedsores; some from exhaustion.

(2) **Acute diffuse myelitis** has a very acute onset with high fever, delirium, rapid development of the myelitic symptoms, and death in a few days.

(3) **Hemorrhagic myelitis** is marked by the sudden intensification of the symptoms of ordinary myelitis.

(4) **Disseminated myelitis** is often subacute.

Treatment.—During the acute stage the most important point is to prevent retention of the urine and infection of the bladder, and bedsores. Later the wasted muscles need massage and electricity.

CHRONIC MYELITIS.

Etiology.—This may follow chronic alcoholism, syphilis, gout, protracted exposure, injury or surrounding inflammation.

Lesion.—The affected part of the cord shows a thick pia. The cord itself is slender, firm, and gray. Microscopically, there are degeneration and disappearance of nerve fibers and cells, excess of neuroglia, thickened blood-vessels, patches of round cells, and secondary degenerations.

Symptoms.—Fever is absent. (*a*) **Transverse thoracic myelitis** is most common. The *earliest symptoms* are paresthesia of the legs, a girdle sensation at the level of the lesion, weakness of the legs and stumbling, difficult micturition. *Later*, symptoms of a transverse lesion, complete or unilateral, slowly develop. (*b*) The lesion may involve the **lumbar cord,** with slowly developing lower-segment paralysis. (*c*) There may be **ascending myelitis** that extends upward to the cervical cord or bulb.

The **course** of chronic myelitis is very slow, lasting years. It may stop at any time with more or less improvement. There may be subacute exacerbations. The chief dangers are infection of the urinary tract or elsewhere and bedsores.

Treatment.—Blisters or cautery to the spine. Hydrotherapy. The general care of health and the prevention of infection are most important.

QUESTIONS.

Give the etiology, lesions, symptoms, and prognosis of acute transverse myelitis.

What are the other forms of acute myelitis?

Give the etiology, lesions, and symptoms of chronic transverse myelitis.

LOCOMOTOR ATAXIA (Posterior Sclerosis; Tabes Dorsalis).

Etiology.—This is a very common nervous disease. Nine-tenths of the cases are in men; usually beginning in the fourth decade; a large majority have had syphilis. Perhaps trauma to the spine or arduous work with exposure may predispose.

Lesions.—This is a *system disease of the afferent tract.* Tabes is supposed to be a primary degeneration of the lower sensory neurons, beginning in the lumbar region. There should be a degeneration of the cells of the posterior root-ganglion in the lumbar region (although sometimes the changes here are less marked than would be expected); secondary degeneration *descending* in the sensory fibers of the peripheral nerve (most apparent in the cutaneous nerves); also *ascending degeneration* of the posterior roots central to the ganglion; and, in the cord, of the column of Lissauer, the nerve fibers in the region of Clarke's column, the column of Burdach in the lumbar and lower thoracic regions, and to a less extent higher up, and the column of Goll throughout its length. The spinal-nerve roots are involved successively as the disease spreads. Upon examining the cord the anterior roots are normal, the posterior are smaller and translucent, the posterior columns of the cord are shrunken and firm, the pia over them may be thick and somewhat opaque. Microscopically, in the degenerated areas mentioned above, there is an excess of neuroglia and an absence of nerve fibers. In certain cases degenerations have been seen in certain cranial nerves and their nuclei, in some cells and fibers of the cerebellum, and some fibers of the cerebrum up to the cortex. Occasionally tabes begins in the cervical region; the changes are precisely analogous, and therefore do not involve the column of Goll.

Symptoms.—First, the various symptoms that make up the clinical picture will be mentioned:

Sensory Symptoms.—(*a*) *Irritative symptoms:* Paresthesias of the arms and legs; the girdle sensation in the trunk; lightning pains, especially in the legs and sometimes very severe; gastric crises (attacks of epigastric pain, often intense, sometimes with vomiting, and recurring for years); other crises—intestinal (diarrhea and colic), rectal (tenesmus), renal (like renal colic without albumin, blood or stones in the urine), also cardiac, bronchial, urethral, clitoral, and laryngeal. (*b*) *Defective sensation:* Cutaneous partial anesthesia, most marked and earliest in the soles, lessening toward the trunk (the patient feels as if walking on cotton). Elective sensory paralysis may be present in very many forms. Sensation is often delayed; the temperature sense perverted; a prick may be felt, but the pain referred to the wrong place (allocheiria) or to two places, and often a sensation persists for a long time. The ulnar nerve is often involved early. Sometimes complete anesthesia of the limbs develops late. *Optic atrophy* with restricted field of vision and dimness of sight is common; it often appears early and may lead to complete blindness. Symptoms like Ménière's disease may appear.

Sensory Disturbances of the Muscular System.—*Ataxia* is very important. It is a loss of coördination, and doubtless a sensory phenomenon. It appears *first in the legs,* and leads to the *tabetic gait*—a straddling, high-stepping, stamping gait; leading to the use of a cane, or later of two canes; it is much worse in the dark, the patient often stumbles and falls. He tires easily, rises from a seat with difficulty, is very uncertain in turning quickly, or in touching any definite point with his toe. Finally the ataxia keeps him in bed. It is present also, to a less extent, in the arms; touching the tip of the nose with a finger, or bringing the two forefingers together can not be accomplished by a quick movement, groping about being necessary; fine movements are affected. The *muscular sense* is impaired. With eyes closed he can not define a passive movement of the limbs; nor can he imitate any position of one limb with its fellow. *Romberg's sign* is very important: when standing with feet together and eyes closed he sways much and may fall. This does not depend on *cutaneous* anesthesia.

Motor Symptoms.—Muscles do not atrophy or change in electric reaction, and even when bedridden from ataxia the muscles are strong. There is lessened tone. Occasionally lower-segment paralysis develops, but it is probably due to a neuritis—at any rate recovery very often follows. This neuritis may be early, and usually affects the musculospiral or peroneal nerves or the eye or the larynx.

Bladder and Rectum.—Constipation is the rule. Difficulty in starting micturition is an early symptom; later there may be retention and liability to cystitis. In very advanced cases there may be incontinence of urine and feces. Impotence is common, often early.

Reflexes.—*Loss of knee-jerk* is constant, very early, and most important. *Argyll-Robertson pupil* (p. 336) is very common and often early. Spinal myosis also is usual. Cutaneous reflexes vary, but often are diminished.

Trophic Symptoms.—They may be wholly absent; or there may be scaly skin, disturbance of hair and nails, petechiæ; perforating ulcer of the foot is not rare, spontaneous fracture may occur. *Charcot joint* (tabetic arthropathy): Usually in the knee, sometimes in other large joints; the joint is much swollen from effusion and great ecchondroses and exostoses in the ends of the bones; the joint surfaces are eroded; the ligaments are stretched and lax, often with abnormal mobility of the joint. The joint is painless.

Cerebral Symptoms.—Tabes and general paralysis of the insane are often associated. Attacks of hemiplegia from hemorrhage, embolism or thrombosis are not rare.

Clinical Course and Stages.—(1) **Preataxic Stage.**—The onset is very insidious, with paresthesia, lightning pains, lasting months or years; then loss of knee-jerk and Argyll-Robertson pupil. Other early, but less constant, symptoms are ptosis, diplopia, defective vision, nyctalopia; gastric or other crises; difficult micturition, perhaps impotence. This stage lasts from a few months to twenty years. (2) **Ataxic stage**: To the above symptoms are added the disturbed gait and muscular sense, with Romberg's sign. Anesthesia may appear. The bladder and trophic symptoms may be more marked. This stage may last for years. (3) **Paralytic stage**: The patient is bedridden from ataxia, the bladder and rectum symptoms are marked; incontinence is common. The mind may fail. The patient dies of exhaustion or pyelonephritis, infection from bedsores, or pneumonia, etc. The disease lasts for years; it may advance steadily or be marked by long periods of intermission and exacerbation. There are very acute cases, lasting only a few months.

The **diagnosis** can be made if the symptoms are looked for. Lightning pains, loss of knee-jerk, and Argyll-Robertson pupil coexisting are diagnostic. Often optic atrophy is early and it is important.

Prognosis.—With optic atrophy the spinal symptoms stop or may ameliorate. The disease often stops in the preataxic stage. In the ataxic stage marked improvement is by no means rare; recovery does not occur, although the disease may stop advancing at any time.

Treatment.—Avoid fatigue and all excesses. Arsenic, strychnin, silver nitrate, and belladonna are recommended. The bladder must be watched and extreme care used in catheterization. Careful gymnastic exercises may reëducate the limbs to a point of great usefulness. There is much symptomatic treatment.

QUESTIONS.

Give the etiology and lesions of locomotor ataxia.

Give the sensory symptoms, the muscular symptoms, the reflexes, the trophic symptoms, and the cerebral symptoms.

Give the course, stages, and prognosis.

Discuss the diagnosis and give the treatment.

ACUTE ANTERIOR POLIOMYELITIS (Infantile Paralysis).

Etiology.—This disease occurs from the second to the fourth year, sometimes later; in small epidemics, or sporadically. It is more common in summer. It is probably an infection.

Lesions.—If this were not an inflammation it would be a system disease of the lower motor-segment. At first there are the lesions of acute hemorrhagic meningitis, involving the anterior horns of the cord in the lumbar and cervical regions, with complete destruction of the ganglion cells in certain segments, and temporary damage by compression of cells in other segments. This region of the cord is supplied by certain branches of the ventral spinal artery, and infectious embolism or thrombosis of these branches is assumed by some authorities. *Later* the affected part of the cord is shrunken and firm; microscopically all or most of the large anterior-horn ganglion cells have disappeared, and there is an excess of neuroglia. The ventral nerve-roots and the peripheral nerves are degenerated and fibrous; the affected muscles are fibrous, fatty, and atrophied.

Symptoms.—Often at the onset there are acute fever (102°–104° F.), delirium, and perhaps convulsions. After two to four days this subsides and paralysis is noted; it reaches its maximum in a few hours or a day or two. In other cases paralysis is the first symptom noted, increasing for from two to five days. The paralysis involves the limbs in any combination, often all the limbs. It is of the lower-segment type. The muscles are flabby, waste rapidly, and in one or two weeks show R. D. The reflexes usually are lost, while sensation and the sphincters escape. In the first month much improvement takes place. After this a little improvement may continue for a few months. The muscles permanently paralyzed are usually the anterior leg muscles of one or both legs; but in other cases the calf muscles; or the deltoid, biceps, brachialis anticus, and supinator longus; or the extensors of the wrist; or the flexors of the wrist. Often there is defective growth and brittleness of the bone. Contractures are not rare.

Prognosis.—Either death or complete recovery is very rare. The permanent paralysis can not be estimated for a month or more, then the electric reactions (p. 330), are a valuable aid.

Treatment.—Ordinary treatment for the febrile stage. *Later*—fresh air and liberal diet. Careful massage of the muscles for months is very important. Electricity, faradic and galvanic (where faradism fails), should be used daily. Strychnin is recommended. Orthopedic surgery is frequently indicated.

Chronic and subacute poliomyelitis in adults does actually occur, but most cases thus diagnosed seem to be neuritis or progressive muscular atrophy.

QUESTION.

Give the etiology, lesions, symptoms, and treatment of acute anterior poliomyelitis.

ACUTE ASCENDING PARALYSIS (Landry's Paralysis).

Etiology.—Usually in males and in the third decade. Etiology and lesions are not understood.

Symptoms.—Rapidly advancing motor paralysis beginning in the legs, spreading to the trunk, then to the arms, shoulders and neck, and finally to the diaphragm and the muscles of swallowing and speaking. The muscles are flabby, but do not waste or change electrically. Reflexes are lost. Sensory and trophic symptoms are absent. Sphincters are normal. There is no fever, but the spleen may be large. They die in a week or less of respiratory paralysis.

Many cases can not be diagnosed from multiple neuritis; in fact, some believe this to be a multiple neuritis. The **prognosis** is hopeless, and **treatment** is vain.

QUESTION.

Give the symptoms and prognosis of acute ascending paralysis.

MYASTHENIA GRAVIS (Asthenic Bulbar Paralysis).

A rare condition of young adults, with unknown etiology and lesion.

Symptoms.—**Myasthenia.**—A muscle in tetanic contraction relaxes from fatigue after a few seconds. If it be rested, it again may contract for a few seconds. This leads to rapid fatigue in ordinary exertion, and in faradic stimulation (myasthenic reaction). All voluntary muscles are involved, first those supplied by the bulbar nerves (eyes, face, mastication, neck). There are no tremors, no wasting, no R. D., no sensory symptoms; the sphincters are normal. The knee-jerks show the same fatigue, disappearing after a few trials and returning after a rest. There are periods of exacerbation and marked remission. The patient may live for several years or only a year or two. Some get well.

Treatment.—Rest, careful feeding, massage, electricity, and strychnin hypodermically in large doses.

QUESTION.

Give the symptoms of myasthenia gravis.

SPASTIC PARALYSIS OF ADULTS.

(Primary Spastic Paraplegia; Primary Lateral Sclerosis.)

Etiology.—The cause is not known. The disease is found in the first half of adult life; sometimes it is a family disease; it may follow syphilis.

The **lesion** is not perfectly constant. A degeneration of the pyramidal fibers at their terminations, or more extensively, is assumed.

Symptoms.—It acts like a system disease of the upper motor-segment. The affected muscles show upper-segment paralysis. The legs slowly grow stiff, especially in the morning; voluntary motion weakens; the reflexes, both deep (knee-jerk, ankle clonus, Babinski) and superficial, are greatly exaggerated; it causes a characteristic gait—the legs are stiff, the thighs adducted, the knees bend but little, the toes are dragged along the ground, sometimes with clonus. Passive movements are more or less resisted. At last the patient is too stiff to walk and is bedridden. *Later*, in some cases similar changes may involve the arms or muscles of swallowing and speaking, or very rarely the sphincters. Sensory pains or paresthesia in the legs rarely appear. The general health is quite good in other respects. Sometimes this is associated with general paralysis of the insane.

Course and Prognosis.—It may last for years without preventing the patient's walking; it rarely shortens life.

Treatment.—Rest, passive motion, arsenic, silver nitrate, potassium iodid and cod-liver oil are recommended.

QUESTION.

Give the etiology, assumed lesions, symptoms, prognosis, and treatment of primary lateral sclerosis.

SPASTIC PARALYSIS OF INFANTS.

(Spastic Diplegia; Birth Palsy.)

Etiology and Lesions.—(*a*) During difficult labor, meningeal hemorrhage on both sides, over the motor areas, is the usual condition, with consequent degeneration of the upper motor-segment. Possibly (*b*) a meningo-encephalitis or (*c*) non-development of the pyramidal tract, especially in premature babies, may be the cause.

Symptoms.—Occasionally, soon after birth there may be convulsions, rigid limbs, and dysphagia. Usually, however, the first thing noted is that the child does not walk when it should. Then examination shows stiff limbs, especially the legs, but often all four limbs (diplegia). There are the ordinary symptoms of upper-segment paralysis. When the child is held so that its toes touch the ground the legs often are crossed from adductor spasm. In time it may walk, but always stiffly and often cross-legged. *Spastic chorea* is common—attempts to grasp an object lead to many jerky movements of hands and fingers. *Bilateral athetosis* also is common—continuous unrest and activity of the muscles; sometimes in head, neck and arms, but most marked in the hands; the fingers are continually flexed, extended, abducted and adducted, separately or together, in most peculiar ways. The movements are increased by excitement; and usually are absent during sleep. Sensory and trophic symptoms are absent; all degrees of mental defect are common.

Prognosis.—Usually the child learns to walk in time. If mentality is good, improvement may be considerable, especially in the head, neck and arms, but less in the legs. The muscles are capable of education by careful and persistent training, and this constitutes the only treatment.

QUESTIONS.

Give the etiology, lesions, and symptoms of spastic paralysis of infants.

Describe spastic chorea and athetosis.

HEREDITARY SPASTIC PARAPLEGIA.

(1) There is a group of cases like the above without mental defect. The lesion is sclerosis of the terminal parts of the pyramidal fibers. (2) Other cases begin in third decade, and progress slowly. They have spastic paralysis, first of legs, later of arms, and finally a distinct atrophic paralysis of muscles and loss of sensation.

AMAUROTIC FAMILY IDIOCY.

A family disease found in Jews, beginning in the first or second year. There are paralysis of the limbs (upper or lower-segment type), optic atrophy, mental symptoms developing into idiocy, marasmus, and death in a year.

PROGRESSIVE SPINAL (CENTRAL) MUSCULAR ATROPHY.

(Chronic Anterior Poliomyelitis; Amyotrophic Lateral Sclerosis; Progressive Bulbar Paralysis.)

Etiology.—Usually in males, beginning after the twenty-fifth year. Perhaps it may follow habitual exposure, worry, or fright. Heredity is negative.

Lesions.—It is a system disease of the entire motor tract, with slow progressive degeneration, which may begin at any level or in several places. In the lower segment there is atrophy of the anterior horn-cells and the motor cranial nerve nuclei with the consequent changes. In the upper segment the short fibers of the cord are involved at first, but, later, degeneration extends upward, more or less, in the pyramidal fibers and may reach and involve the cortical cells in the motor area.

Symptoms.—(1) **Aran-Duchenne Type.**—The lesion is chiefly in the lower segment. *Sensory symptoms* are absent except for some slight pain or paresthesia. *Motor symptoms:* There are very slowly advancing weakness, atrophy, change of electric reaction and weakening reflexes in the muscles, attacking them in a fairly regular order. First, all the muscles in the metacarpus atrophy, leaving apparently only skin and bone and tendon; the central phalanges are strongly extended, the others flexed (claw hands). Next the forearm atrophies; then the deltoid, causing a flat shoulder and often sagging of the humerus from the glenoid cavity; then the other arm muscles, the triceps being the last. It then attacks the trunk, with lordosis, and perhaps the neck, with unsteady head. Still later the legs also may show the same changes. "Living skeleton" freaks have this disease. Sometimes the cranial nerve nuclei degenerate, adding the symptoms of bulbar paralysis, but the platysma, face, and upper part of the trapezius escape. Fibrillary twitching is present. Complete R. D. is found only when atrophy is practically complete. Contractures may develop. Sphincters are normal. The *course* is very slow, lasting for many years, and death is due to some intercurrent disease or to bulbar paralysis.

(2) **Amyotrophic Lateral Sclerosis.**—The lesion in the upper segment predominates, at least at first. The paralysis is more or less spastic and attacks the muscles in the order given above. In the arms this gradually changes to the atrophic type, but in the legs usually remains spastic. The bulb may be involved.

(3) **Bulbar paralysis** may extend to the lower levels or may develop by extension from lower levels. For its symptoms see p. 382.

The **course** of the whole disease is slowly progressive and hopeless, but living skeletons may live several years.

Treatment.—Electricity, massage, gymnastics, and strychnin hypodermically, gr. $\frac{1}{100}$ to $\frac{1}{15}$ daily, are used.

QUESTIONS.

Give the etiology, lesions, and types of progressive spinal muscular atrophy.

Give the symptoms of each type and the prognosis.

PROGRESSIVE NEURAL MUSCULAR ATROPHY.

(Peroneal Type of Progressive Muscular Atrophy.)

A very slow atrophic paralysis of the muscles with fibrillary contractions. It differs from the spinal form in the following particulars: It is a family or hereditary disease, beginning in the second decade. It involves first the peroneal *muscles and* the muscles in the feet, causing double acquired pes equinus or equinovarus; spreads to thighs, calves, trunk, and after some years to the hands and arms (very rarely these are attacked first). *Sensory symptoms* are severe pains, paresthesia, anesthesia. Electric reactions change—lessened excitability of nerve and muscle, rarely R. D.; this is found in apparently normal muscles as well as in the atrophied muscles. The disease does not shorten life, but the patients do not get well.

Treatment as in the previous forms.

QUESTIONS.

Give the etiology, symptoms, and prognosis of progressive neural muscular atrophy.

Give the differential diagnosis from the previous disease.

ATAXIC PARAPLEGIA.

(Combined Posterior and Lateral Sclerosis.)

Etiology.—More often in males, possibly related to exposure or trauma, but not to syphilis.

Lesion.—The *posterior sclerosis* differs from that of tabes as follows: It is not marked in the posterior-root zone, nor most marked in the lumbar cord and column of Goll. The *lateral sclerosis* is not confined to the crossed pyramidal tract, but involves the direct cerebellar and the anterior pyramidal tracts also.

Symptoms.—(1) A slow development of **ataxia** (difficulty in walking, especially in the dark, Romberg's sign is usually present) associated with (2) **spastic paraplegia** (the spastic gait less marked than in primary lateral sclerosis, increased knee-jerk and often ankle clonus). (3) the **sphincters** are often involved, but *sensory* and *eye symptoms* are absent. It may end as general paralysis of the insane.

The **prognosis** is worse than that of primary lateral sclerosis because of the danger of urinary sepsis.

The **treatment** is like that of primary lateral sclerosis.

QUESTION.

Give the differential diagnosis between ataxic paraplegia and locomotor ataxia; between ataxic paraplegia and primary lateral sclerosis.

HEREDITARY (FRIEDREICH'S) ATAXIA.

Etiology.—A family disease that usually appears before the twelfth year, but sometimes in adolescence; it may follow acute infectious diseases, or trauma, or exposure.

Lesion.—The cord is smaller. There is sclerosis of the posterior columns, most marked in the lumbar region and column of Goll; also sclerosis of pyramidal tracts and direct cerebellar tract, and of Clarke's column. Perhaps it is a primary gliosis with subsequent degenerations.

Symptoms.—It begins in childhood. There are ataxia, first in the legs, with stumbling, uncertain gait; Romberg's symptom, Babinski's sign, early loss of knee-jerk, and often pes cavus. Later, ataxia in the arms and often oscillatory movements of the head and trunk, and scoliosis. The big toe often is kept in dorsal extension. There may be nystagmus, often stuttering and other disturbances of speech. Afterward there are menstrual disturbances in the female and impotence in the males. Other tabetic symptoms are absent, such as Argyll-Robertson pupil, pains, etc. It is progressive and incurable, but does not kill.

Treatment.—Orthopedic treatment often helps.

QUESTIONS.

Give the etiology, lesions, and symptoms of hereditary ataxia.
Give the differential diagnosis from locomotor ataxia.

Syphilitic spinal paralysis (Erb) follows syphilis, and therefore is a disease of adults. There is a primary sclerosis of the pyramidal, direct cerebellar, Gowers, Goll, and Burdach columns. The **symptoms** are those of spastic paraplegia, bladder symptoms, retention, and incontinence (retention of urine may be the first symptom); impotence, incontinence of feces, paresthesia, and the girdle sensation. They may improve somewhat, but do not wholly disappear. The **treatment** is antisyphilitic.

Primary combined sclerosis (Putnam) is almost always found in debilitated women. There are rather large patches of degeneration throughout the posterior and lateral columns in the thoracic and cervical regions. **Symptoms:** Paresthesias; a somewhat spastic paraplegia, later becoming atrophic; the arms are somewhat paralyzed; mental deterioration may follow. Profound anemia or cachexia may precede or follow the nervous symptoms.

Hysteric spastic paraplegia may be an excellent imitation of organic disease, but the loss of power is usually moderate, the deep reflexes persist and the gait is not spastic.

Hereditary cerebellar ataxia differs from Friedreich's ataxia in appearing in adults, in sometimes showing sclerosis in the cerebellum, in persistence of knee-jerks, and finally in a spastic condition of the legs without clubfoot or sclerosis. Optic atrophy may be present or absent.

Ergotism, lathyrism and **pellagra** show sclerosis of the cord.

Pernicious anemia or **other cachexias** may have posterior and lateral sclerosis with symptoms resembling Putnam's type.

Ophthalmoplegia (see p. 338) is sometimes a system disease of the lower motor-segment.

CAISSON DISEASE.

The **cause** is too abrupt transition from air compressed to three atmospheres or more, to air at atmospheric pressure.

The **lesion** is supposed to be due to bubbles of free gas, rendered insoluble in blood and lymph by the decrease of pressure. The spinal cord shows fissures from laceration. Some long-standing cases have shown myelitis also.

Symptoms may develop at once or after a few hours. They are: (1) **Pains** (the "Bends") in the knees and other large joints, often intense; perhaps they may involve the muscles also, or the epigastric region, sometimes with vomiting. (2) **Paralyses** are less common—*paraplegia* is the usual form. It involves motion, sensation, and the sphincters. It may last a few hours only or for days or weeks. The worst cases become comatose and die in a few hours.

The **prognosis** varies with the severity, but fatal cases are few.

Treatment.—Preventive: Slow decompression. **Cura-**

tive: The patient should be restored to compressed air at once and the pressure then reduced very slowly. Special air-tight wards should be provided for this wherever caisson work is done.

QUESTION.

Give the etiology, lesions, symptoms, and treatment of caisson disease.

DISEASES OF THE MEDULLA OBLONGATA.

CHRONIC BULBAR PARALYSIS.

(Glossolabiolaryngeal Paralysis.)

Etiology.—More common in males; rare before forty.

Lesions.—It may be a late or an early lesion of progressive spinal muscular atrophy, of which it really is only a part. Sometimes the bulbar part of the lower motor-segment is involved alone. There are degeneration and atrophy of the motor cranial nerve-nuclei in the medulla (hypoglossal, spinal accessory and glossopharyngeal) with the consequent degeneration and atrophy of nerves and muscles.

Symptoms.—(Review the paralyses of above nerves and progressive spinal muscular atrophy.) The onset is very slow, the order of involvement being (1) the **tongue**—disturbance of speech, of the forming and swallowing of the bolus of food, of voluntary movement; also the presence of fibrillary twitchings; (2) **lips**—disturbance of speech (*p*, *b*, *f*, *m*, *w*, *v* sounds), of drinking and whistling; the mouth can not be kept shut, and the patient drules; (3) **palate and pharynx**—interference with palatal sounds (*k*, *g*, *q*), and regurgitation of liquids through the nose; (4) **larynx**—aphonia, dyspnea, modified cough, etc. There are no sensory symptoms. The patient often becomes extremely emotional.

The **course** is slow, and death is due to marasmus, aspiration pneumonia or, rarely, to paralysis of the respiratory center. Of ocurse, there are often the other symptoms of progressive muscular atrophy and the reactions, etc., of paralysis of the above nerves.

Treatment.—The most important point is gavage, to avoid aspiration pneumonia and starvation.

OTHER BULBAR PARALYSES.

Acute apoplectiform bulbar paralysis is due to hemorrhage, embolism or thrombosis of one or both vertebral arteries in arterial disease, especially syphilitic. The onset is very abrupt, perhaps with headache and vertigo, but very soon there is a sudden paralysis of the bulbar nerves, less symmetric and more irregular than in the chronic form. Often upper-segment paralyses of lower levels develop. The patient may die in a few hours or days, or the paralysis may become less extensive, but some muscles will be found atrophied.

Acute inflammatory bulbar paralysis (Myelitis bulbæ, acute inferior polio-encephalitis) is an acute myelitis involving the nuclei of the bulbar nerves, like anterior poliomyelitis in the cord. It is rare. The onset is a little less acute than the apoplectiform type and may be associated with fever.

Pseudobulbar paralysis is due to a bilateral lesion of the upper motor-segment, usually a cortical thrombosis, rarely at the knee of the internal capsule. The paralyses are not perfectly symmetric and do not appear at exactly the same time on both sides. It has the characteristics of an upper-segment lesion.

QUESTIONS.

Give the etiology, lesions, symptoms, and treatment of chronic bulbar paralysis.

Discuss other bulbar paralyses.

DISEASES OF THE BRAIN.

CEREBRAL LOCALIZATION.

General or *diffuse cerebral symptoms* may be present with any brain lesion. *Local* or *focal symptoms* may locate the site of a lesion. The *clinical course* of the disease may suggest the nature of the lesion.

General or Diffuse Cerebral Symptoms.—Headache, vertigo, vomiting, disorders of consciousness, general convulsions, choked disk, tinnitus, changes in the rate and rhythm of heart and breathing, etc.

Local or Focal Symptoms.—Lesions in many parts of the brain cause no focal symptoms; these are "silent regions" and they still include most of the brain.

Of the focal symptoms some are due to so serious a lesion of the brain tissue that it is permanently destroyed. These are called **direct symptoms** or symptoms of deficiency.

Others are due to physiologic disturbance without destruction of tissue, and are called **indirect** or **remote symptoms.** They are due to pressure, circulatory disturbances, etc. They involve a considerable area surrounding the destroyed tissue, and sometimes occur even in remote parts of the brain. If these parts regain their functions the symptoms disappear, perhaps in a few hours or days, or not for a month or two.

Cortex.—A lesion of the pyramidal cells of the **motor area** (p. 322) causes focal symptoms. **Irritation** results in muscular twitching and convulsions. Clonic convulsions often begin in a certain muscle and spread to others on that side of the body or even to the other side. This is called **Jacksonian** or **cortical epilepsy.** The muscles in which the clonus starts give the direct symptoms and locate the lesion; the others give indirect symptoms. The order of involvement shows that the irritation spreads from its starting-point to successively contiguous portions of the cortex, and there are no immune intervening centers. Consciousness usually persists. **Paralytic symptoms** have the characteristics of upper-segment paralyses. Monoplegia of upper-segment type is usually caused by a small cortical lesion. If the paralysis begins at one point and *gradually* spreads to a hemiplegia it is usually cortical, and it spreads to contiguous parts of the cortex. Often cortical paralysis is associated with irritative disturbance (convulsions) corresponding to surrounding areas of the cortex. Lesions of Broca's convolution are important and will be mentioned with Aphasia.

Parietal Lobe.—Besides containing part of the motor area, this seems to control muscular sense, and destructive lesions in it may cause loss of muscular sense. The above parts of the cortex (except the motor-speech area) affect peripheral structures on the other side of the body only.

Temporal Lobe.—The upper part of the left superior temporal convolution is related to the auditory nerve, and a lesion here results in word deafness.

Occipital Lobe.—A lesion of the cuneus and first occipital convolution causes homonomous lateral hemianopia of the same side of the retinæ, and hence of the other side of the fields of vision.

Centrum Ovale.—Lesions of the association and commissural fibers are not important; those of the **projection fibers** cause the same symptoms as lesions of the corresponding part of the cortex. They are, however, rare.

Internal Capsule.—The anterior limb is a silent region. The knee and anterior two-thirds of the posterior limb carry pyramidal fibers; the posterior part of this limb carries sensory fibers. Lesions here are usually destructive (hemorrhage), and the fibers are so compact that complete hemiplegia of the other side of the body usually results, except in the orbicularis palpebrarum and frontalis, which escape. If the posterior fibers are involved there is complete sensory paralysis of the other side of the body (complete anesthesia, deafness, hemianopia, etc.).

Cerebral Peduncles (Crura).—Lesion here causes **lower-segment paralysis** of the oculomotor nerve of the same side and **upper-segment paralysis** of the lower-level muscles of the other side (cerebral hemiplegia with alternate (crossed) oculomotor paralysis, or "the syndrome of Weber"). This and similar combinations of paralyses, partly on one side, partly on the other, are called crossed or alternate paralyses; the paralysis on the same side as the lesion is of the lower-segment type. If the **tegmentum** is involved, there is hemianesthesia of the other side of the body.

Pons.—The characteristic symptoms are cerebral hemiplegia on the other side of the body with alternate facial paralysis (involving eyelid and forehead also) on the same side. The fifth or sixth nerves may be paralyzed, and if the sixth nucleus is involved there is conjugate deviation of both eyes away from the lesion. **Bilateral disease** gives a double, crossed paralysis, and if acute may cause marked rigidity of the limbs, myosis, and hyperpyrexia, perhaps with trismus, rapid pulse, and glycosuria.

The **optic thalamus** is rather obscure, some of the points were given under the Optic Tract.

The **cerebellum** also is very obscure. Probably a lesion of the worm causes vertigo and a staggering, drunken gait (disturbances of equilibrium).

Recapitulation of some of the above points: (1) The usual cause of ordinary hemiplegia is a lesion of the internal capsule. (2) Monoplegia of upper-segment type is usually due to a lesion of the cortex at the corresponding point of the motor area. (3) Hemiplegia or monoplegia associated with clonic convulsions of part or all of that side of the body is almost always cortical. (4) Epileptiform convulsions limited to a few muscles, a limb, or one-half of the body, are almost always cortical. (5) Hemiplegia with alternate oculomotor paralysis means a lesion in the crus. (6) Hemiplegia with alternate facial paralysis means a lesion in the pons. (7) Complete hemianesthesia, involving special senses also, is usually due to a lesion in the posterior part of the internal capsule. (8) Staggering and vertigo may suggest a cerebellar lesion.

QUESTIONS.

Mention the diffuse cerebral symptoms.

What are direct symptoms? Remote symptoms?

What are the irritative and the paralytic symptoms of the motor area?

What is Jacksonian epilepsy, and where is the lesion?

Where is the usual lesion in monoplegia; in hemiplegia?

What symptoms are due to destructive lesions of the parietal lobe, the temporal lobe, the occipital lobe, the centrum ovale, the various parts of the internal capsule, the crus, the tegmentum, the pons and the cerebellum?

Review the more important points given in the recapitulation.

APHASIA AND SOME ASSOCIATED CONDITIONS.

The cortical speech centers are: (1) **Motor,** in the third left frontal gyrus, near the hypoglossal and facial areas; (2) **Sensory**—(*a*) *Auditory speech center* (word hearing) in the posterior half of the first temporal gyrus at the eighth-nerve cortical area; (*b*) *visual speech center* (word seeing) in the angular gyrus of the occipital lobe, near the eye center. (*c*) Some add another motor center—*the word-writing center,* near the area for the hand. In right-handed persons these are on the left side of the brain; in left-handed persons, on the right side of the brain.

Aphasia is due to destructive lesions in one or more of these centers or in the communications between them. It is the loss of the ability to (1) use words intelligently in speaking or writing, or both; or (2) to recognize spoken or written words; or (3) to recall words. It may involve only a few words or the entire vocabulary. Some of the many subdivisions are:

(1) **Motor Aphasia.**—The *lesion* is in Broca's convolution. It is impossible to properly utter words spontaneously or to repeat them after another person. In the mildest cases only a few letters may be confused; in the severest cases, perhaps, no word can be spoken, merely senseless sounds.

(2) **Sensory aphasia** (word deafness) follows a lesion of the word-hearing center. Words uttered by others seem to be heard as sounds with no conception of their meaning.

(3) **Amnesic Aphasia.**—When a familiar object is presented to a patient he may name it, but he can not afterward recall the word to utter it spontaneously. He may fail to recall even his own name, but may recognize it when he hears it.

There is any number of other subdivisions of aphasia. Some of the other terms used are *paraphasia*, the use of wrong words; *agraphia*, also motor, an inability to write words; *alexia*, word-blindness, a sensory form, with inability to recognize written or printed words; *echolalia*, the senseless repetition of one or a few sounds or words; *total aphasia*, all kinds combined.

The **cause of aphasia** is usually thrombosis or embolism, less often hemorrhage of the branches of the middle cerebral artery that go to the cortical speech centers; usually it is part of a right hemiplegia. Without motor paralysis it may be due to small tumors or abscesses, local meningitis, or trauma. Hemorrhage into the internal capsule may cause transient aphasia. It may occur with uremia, hysteria, epilepsy, etc.

Prognosis.—In children the other side of the cerebrum may be taught to direct speech satisfactorily, but in adults this is possible to only a slight degree.

Treatment.—Careful persistent attempts to educate the centers of the opposite side. Occasionally the cause may be amenable to treatment.

QUESTIONS.

Define aphasia, alexia, agraphia, echolalia, motor aphasia, sensory aphasia, amnesic aphasia, and paraphasia.

Where are the speech centers?

What are the causes of aphasia, the prognosis, and treatment?

CEREBRAL MENINGES.

Leptomeningitis has been described on p. 55.

PACHYMENINGITIS.

External pachymeningitis extends from caries of bone, usually in middle-ear disease or syphilis. Pus collects between the dura and the bone. The symptoms are indefinite, but symptoms of cerebral pressure may be present.

HEMORRHAGIC PACHYMENINGITIS (Hematoma of Dura Mater).

Etiology.—It occurs in old men with chronic insanity or chronic alcoholism; possibly may follow the acute fevers, hemorrhagic diseases, or profound anemia. It is common in asylums; very rare elsewhere.

Lesions.—A thin, fibrous membrane, very rich in blood-vessels, may be attached to the inner surface of the dura. These vessels may rupture and a clot forms. There may be clots with no vascular membrane. The clot may be laminated and ¼ in. thick, or it may soften and form a cyst. Often there is atrophy of the convolutions.

Symptoms may be absent or there may be headache or cortical paralysis, apoplectiform seizures, convulsions, hemiplegia.

The **diagnosis** is usually made at the autopsy. There is no special treatment.

QUESTION.

Give the causes, lesions, and symptoms of hemorrhagic pachymeningitis.

CIRCULATORY DISTURBANCES OF THE BRAIN.

Anemia.—Etiology.—(1) *Part of a general anemia:* (*a*) Profuse hemorrhage from any source. (*b*) Profound anemia from any cause. (2) *Local anemia of the whole brain:* (*a*) Cardiac weakness from any cause, particularly in the erect position. (*b*) Aortic stenosis. (*c*) Spasm of the cerebral arteries from emotion or, perhaps, with arterial disease. (*d*) Sudden accumulation of the blood elsewhere; especially in the abdominal vessels in shock or after too abrupt removal of ascites or other cause of pressure; or after a very large stool, after long constipation, with sudden filling of the intestinal vessels (emotion is often an important factor in these cases also). (*e*) Increased intracranial pressure may squeeze the blood out of the brain, as in hydrocephalus or meningitis. (3) *Local anemia of parts of the brain,* due to pressure from tumors, clots, abscesses or to embolism or thrombosis. This last group is not discussed here.

Lesions.—The vessels of the meninges and the brain are empty, except the largest veins and the sinuses. The brain is pale, often firm.

Symptoms.—*Acute Anemia.*—The most typical cases seem to follow spasm of the arteries. There may be precordial oppression, palpitation, pallor, yawning, spots and swimming before the eyes, tinnitus, then loss of sight and hearing, myosis, a feeling of weakness, and within a few seconds the patient falls unconscious. The unconsciousness lasts a few seconds or minutes. The shorter, simpler attacks are syncope (fainting).

Subacute anemia, as in a large hemorrhage, shows the same symptoms with dyspnea, often twitchings or convulsions, but they develop less abruptly.

Chronic anemia has irritative and paralytic symptoms. There are vertigo, headache, sleeplessness or restless sleep, spots before the eyes, palpitation, occasionally low delirium, paresthesia, perhaps clonic convulsions and a tendency to faint. The symptoms are more marked in the upright position.

The **prognosis** is that of the cause but with inanition; syncope may be fatal.

Treatment.—The horizontal position or elevation of the legs and hips. In simple fainting, diffusable stimulants—ammonia, alcohol, ether; or vasodilators—the nitrites and counter irritations are used. With loss of blood, after the bleeding has stopped, blood may be forced to the head by gravity and by bandaging the limbs upward. Restore the volume of blood by infusions, enemata and transfusion. Anemias need their proper treatment. Wasting discharges need a nutritious diet.

The **hydrencephaloid condition** may occur in babies, especially during the first half-year, with protracted and exhausting diarrhea. It is a form of cerebral anemia. The child is shrivelled, the fontanelles depressed, there is a semicomatose condition, with myosis, open-staring eyes, perhaps convulsions, a rapid small pulse; and, later, coma, big pupils, strabismus, retracted head, and death.

Hyperemia.—(*A*) *Active hyperemia* may be due to (1) hypertrophy of the left heart; (2) plethora; (3) to mental or physical exertion; (4) the inverted position; (5) a heavy meal; (6) alcohol, etc.; (7) possibly to vasomotor paralysis with contraction of the superficial vessels, after chilling, etc. (*B*) *Passive hyperemia* is found in (1) general venous congestion from any cause; (2) asphyxia; (3) pressure on the superior vena cava, or veins from the head; (4) thrombosis of the jugulars or cerebral sinuses.

Lesion.—Active hyperemia is not known post mortem. In passive hyperemia all the vessels may be distended, and there is an excessive amount of blood in the cranium. Perhaps this blood fills so much space and so lessen the flows that the brain cells get too little blood, and the real condition is equivalent to anemia.

Symptoms.—There is no agreement as to the symptoms of congestion of the brain. Perhaps *active hyperemia* may lead to headache, fulness and throbbing in the head, tinnitus, dizziness, a red face, sleeplessness, mental confusion. These symptoms may be constant, or occur in attacks of variable duration. *Obstruction to the veins* may be associated with dulness, torpor, and coma.

Treatment.—Elevate the head, apply ice, use venesection, purging, and causal therapy.

Cerebral edema may be found with atrophy of the convolutions, obstruction of veins, in nephritis, and with edema elsewhere; probably it is often an antemortem change. It is found locally around tumors and abscesses. The **lesions** (wet brain): There is an excess of liquid in the meninges and ventricles, and sometimes the cut surface of the brain itself looks wet. The symptoms are not known.

CEREBRAL HEMORRHAGE.

Apoplexy is used strictly to denote hemorrhage in brain tissue, loosely to denote other vascular disturbances of the brain or meninges, causing similar symptoms. **Intracranial** or **cerebral hemorrhage** includes (1) hemorrhage into the brain itself, (2) meningeal, and (3) ventricular hemorrhage.

Etiology.—The *predisposing factor* is arteriosclerosis with its etiology (p. 266). The *exciting cause* is a sudden increase of pressure, perhaps due to exertion, emotion, overeating, alcohol, or to no discoverable cause. It is more common in thick-set, short-necked, red-faced persons, and usually after fifty. Less often cerebral hemorrhage occurs in younger persons, associated with disease of the arteries, or with hemorrhagic conditions from any cause, such as leukemia, anemia, purpura, and certain infections. It may be due to excessive blood pressure in a paroxysm of whooping-cough, etc, ; or to trauma.

Lesions.—The diseased artery usually shows *miliary aneurysms,* one or more of which have ruptured. **Hemorrhage into the internal capsule**: (Sixty per cent. of all cases of cerebral hemorrhage). A lenticulostriate artery of the left side (a branch of the middle cerebral to the internal capsule, called the artery of cerebral hemorrhage) ruptures as above. The clot forms and lacerates the brain tissue, with the formation of a cavity, lined with ragged bloody brain tissue. If small, the clot may be limited to the internal capsule, the lenticulate nucleus and the optic thalamus. If larger, the cortex of that side may bulge and the convolutions look flattened and may be soft; the clot may invade the centrum ovale or burst into the lateral ventricle, and perhaps spread to other ventricles. If the clot is small, *repair* may take place. There is absorption of the clot and necrotic brain tissue, leaving a brown, then a yellowish, discoloration. This area becomes encapsulated with fibrous tissue, with the formation of a scar often pigmented, or of a cyst with clear yellowish contents. Secondary degeneration of the fibers destroyed (pyramidal fibers) occurs. **Other forms** of intracerebral hemorrhage are less common. (*a*) *Meningeal* hemorrhage may be extradural, usually from a fractured skull; or within the dura, generally in the pia-arachnoid and from a branch of a Sylvian artery. The clot may flatten the convolutions or lacerate them. Finally, moderate-sized clots are well absorbed, but new connective tissue or even a cyst results. With destruction of brain cells there is secondary degeneration. (*b*) A *cerebellar* hemorrhage may occur. (*c*) *Ventricular hemorrhage* is rarely primary. It usually follows the laceration of the brain in other intracerebral hemorrhage. Blood may fill one ventricle or even all of them. The convolutions look flattened.

Symptoms.—Rarely *prodromal* symptoms, called "warnings," may be present for a few days or weeks, such as fulness in the head, vertigo, excitability.

(1) **The Primary Symptoms.**—(*A*) *The Apoplectic Stroke.*—With or without some muscular strain or emotion there may be for a few seconds vertigo, disturbed speech, or possibly weakness on one side; then the patient falls in coma. The face is dusky, the heart is slow and forcible, the pulse hard; respiration stertorous, slow, irregular, often Cheyne-Stokes; the cheeks blow out with expiration, the lips are flabby, more so on one side; temperature is normal or subnormal. **Nervous symptoms:** Complete coma. The patient can not be aroused. It may be impossible to determine any paralysis at this time, but in other cases there may be complete loss of muscular tone on the paralyzed side only, shown by greater flabbiness of the lip and cheek of that side; by conjugate deviation of the head and eyes, usually toward the side of the lesion; and also the limbs if raised drop at once, but on the other side they drop slowly. The cutaneous and deep reflexes are usually abolished everywhere, but cutaneous reflexes may persist on the unaffected side if the stimulus is strong and persistent—*e. g.*, a deep and protracted prick with a pin. The pupils vary and are often unequal. The light reflex is usually lost, but occasionally it may be present

but very sluggish. Feces and urine may be passed involuntarily or retained. The urine may be albuminous, may possibly contain sugar, and often is increased in amount.

Variations in this Stage.—Onset: Coma may take several minutes to develop. Perhaps the hemiplegia may precede it; rarely convulsions occur. In *very severe cases* the face is ashy pale; the pulse very small and feeble; the heart weak and rapid; all muscles are completely relaxed. These cases are apt to be fatal. The patients may die in a few minutes, or after a day or two, with no lessening of the depth of the coma, or perhaps not for a week. *Ingravescent apoplexy:* The general brain symptoms develop gradually, stupor increases, and coma may be delayed several hours. This is rare with hemorrhage into the internal capsule, but is common with cortical hemorrhage. Rarely a small hemorrhage causes hemiplegia without coma.

(*B*) The *reaction* sets in after a couple of days with fever (101°–103° F.) that may last one to three weeks. The coma may last a few hours or several days; it may go rather quickly or more gradually, and may relapse. It may be followed by restlessness, mental confusion or delirium. *Early rigidity*, a marked spastic condition of paralyzed muscles, may appear now. Trophic symptoms occasionally occur, especially rapid extensive sloughing at the lower part of the back and on the paralyzed side. Fine râles at the bases of the lungs are common. When consciousness returns in the reaction, the *paralysis* becomes evident. It is usually a *hemiplegia.* When complete this involves all the skeletal muscles below the eyelids on one side of the body, including half the tongue. Paralysis is shown by loss of voluntary movement and by flabbiness. In the face the affected cheek bellies out in expiration, the affected halves of the lips can not be controlled. Usually emotional expression is less affected than deliberate voluntary movements. The tongue, when protruded, is pushed toward the paralyzed side, speech is thick and defective in all cases, and with right hemiplegia (in a right-handed person) aphasia may be present. The shoulder and arm are more completely paralyzed than the leg. The intercostals and abdominal muscles may be somewhat weakened. The sphincters are usually normal, but retention may continue for a while. The *reflexes* are lost during the coma. They return after consciousness does, usually first on the sound side, but, perhaps, not for a considerable time (weeks) on the paralyzed side. When, however, they do return, they are much exaggerated on this side. The electric reactions do not change. Sensory disturbances are usually wholly absent or slight, but rarely hemianesthesia is seen.

(*C*) In time the *paralysis usually lessens* to a marked extent, especially in certain muscles. The leg improves most, and the greatest improvement is in the extensors of the knee and the plantar flexors of the ankle. The resulting extension of the knee and ankle gives the characteristic gait —the knee is stiff, the foot in walking is swung around in a semicircle with the inner side of the front of the foot and toes dragging along the ground. The arm improves less than the leg; and the improvement is most marked in the flexors and pronators of the fingers, wrist, and forearm, in the flexors of the elbow and in the adductors of the humerus. As a result the fingers are clinched over the flexed thumb, the wrist somewhat flexed, the forearm half-pronated and flexed, the arm held close to the trunk. The face often improves rather quickly. These changes appear within two months or so. Passive movements in the limbs are perfectly possible in this stage.

(2) **The Chronic or Secondary Symptoms.**—Improvement does not continue indefinitely, but the paralyzed muscles get rigid and hard and tend to shorten. The stronger ones (which happen to be the muscles that are more apt to recover) overpower the others, and in time the limbs are fixed in the positions mentioned. Passive movement is practically impossible, *contractures* have developed and are more marked in the arm than in the leg. The electric reactions are not changed, the reflexes are greatly increased. Trophic disturbances may occur—the red, shiny, cold skin, especially in the arms, also arthropathy, tremors, spastic (posthemiplegic) chorea, and athetosis (see p. 376). Rarely the muscles atrophy, but the cause is really not known. Mental irritability and emotional instability are common sequels.

Course and Prognosis.—The patient may die suddenly or at any time within a few days without regaining consciousness. He may relapse into coma and die; or he may have another stroke after any interval. Edema of the lungs or pneumonia may kill.

Special Forms of Cerebral Hemorrhage.—The characteristic symptoms of hemorrhage into the crus, the pons, the cortex are mentioned on p. 384. Furthermore, cortical hemorrhage often shows great improvement, even complete recovery, especially when traumatic (except in infants, see p. 376). Hemorrhage into the ventricles is quickly fatal, often with convulsions, high temperature and slow pulse. Hemorrhage into the internal capsule usually leaves permanent hemiplegia.

Treatment.—*At first*, if the skin is dusky and the pulse slow and full, raise the head and bleed the patient (1 pt.), put ice to the head, purge with croton oil, gtt. ij, or calomel, gr. v–x. Later the bladder and bowels must be looked after carefully. The diet should be liquid. Gavage is often needed. The weak fast hearts, with pale skin, need stimulation.

Subsequent treatment is most important—electricity, massage, passive movements, and attempts to reëducate the muscles may prevent contractures. When fully developed they can not be cured. The causative factors should receive attention.

EMBOLISM AND THROMBOSIS OF THE ARTERIES.

(Cerebral Softening.)

Etiology.—**Embolism.**—Emboli may be dislodged from (*a*) the heart with endocarditis, mitral or aortic; or (*b*) occasionally from the aorta in atheroma or aneurysm; or (*c*) from the carotid arteries; or (*d*) the lungs, especially in gangrene and putrid bronchitis; (*e*) it may occur in the puerperium. **Thrombosis** occurs (*a*) with arteritis of the cerebral artery, especially syphilitic, associated with feeble heart action. (*b*) Rarely it may occur in marantic conditions with anemia.

Lesions.—**Embolism.**—There is first the source of the embolus. The embolus usually lodges in the Sylvian artery or its branches and on the left side. The area supplied by the plugged artery may be white or red (from regurgitation of blood). In a day or two it softens and necroses, may be almost liquid. The glia cells are fatty and granular, the ganglion cells and fibers are granular. The subsequent changes are like those that follow hemorrhage. Multiple foci may be present after repeated attacks. **Thrombosis** shows the same changes, but the arteries are already diseased. The thrombus is usually in the Sylvian artery or the basilar artery.

Symptoms.—The differentiation from hemorrhage is seldom correctly made, but the following points are suggestive: **Embolism**: The existence of heart disease, the total absence of premonitory symptoms, the very abrupt onset; later, aphasia is more common (with right hemiplegia); and less extensive paralyses are more common. Improvement is often marked. **Thrombosis**: There are the symptoms of the cause (arteritis, syphilis, weak heart); premonitory symptoms are very common; coma may be absent, even with extensive paralysis; the paralysis is more apt to begin in a few muscles and spread gradually, becoming a hemiplegia only after several hours or a day or two. There may, however, be no symptoms, even with an extensive thrombosis.

Treatment.—Causative, especially antisyphilitic. In the attack the patient should be placed horizontally, and often needs stimulation, otherwise treat like hemorrhage.

LARGER ANEURYSMS OF THE CEREBRAL ARTERIES.

Aneurysms of the brain, larger than miliary size, are single or multiple. Usually the only symptoms are those of a fatal apoplexy when the aneurysm ruptures. The larger ones, an inch or more in diameter, may give the symptoms of tumor. The diagnosis is made at the autopsy.

CEREBRAL ENDARTERITIS.

This is very common as ordinary arteriosclerosis and also as a special syphilitic form (nodular periarteritis). The symptoms are given in part on page 268, and in part with the preceding diseases of this section.

THROMBOSIS OF THE CEREBRAL VEINS AND SINUSES.

(*A*) **Primary** or **marasmic thrombosis** is rare. It is due to feeble circulation or increased coagulability of the *blood, or both.* It may occur (1) in the first half-year of life with severe and protracted diarrhea. (2) Later, with chlorosis or anemia, usually in the superior longitudinal sinus and associated with thrombi in other parts of the body. This is called autochthonous sinus thrombosis. (3) Marantic thrombosis is found also in cachexias of cancer, phthisis, etc., involving the sinuses and veins. (*B*) **Secondary** or **infectious thrombosis** usually follows extension of inflammation, especially from the internal or middle ear, injury to the skull, meningitis, erysipelas of the head, or mastoiditis. Suppuration usually follows, and involves the lateral and superior petrosal sinuses. The **symptoms** vary much. In *cachexias* there may be headache, delirium, stupor, muscular rigidity, convulsions, coma, death. In *infectious cases* there is sepsis also. It is seldom correctly diagnosed, but where the cause exists and the head symptoms appear it may be suspected. The infectious cases are often saved by the surgeon.

INFANTILE HEMIPLEGIA.

Etiology.—This occurs in the first two years, usually without obvious cause; it may follow measles, or scarlatina; or possibly injury from instrumental delivery.

Lesions.—(1) **Atrophic sclerosis** is the most common. It involves groups of convolutions, an entire lobe or one hemisphere. The meninges are thickened and adherent, the brain is hard and smooth, the ventricle dilated. The primary cause is unknown, possibly it is thrombosis of the veins. (2) **Porencephalus**: Cavities and cysts form in the arachnoid or brain tissue, or involve the ventricles on one side of the brain. It may be a developmental defect or follow hemorrhage at birth. (3) **Embolism, thrombosis or hemorrhage** sometimes is found. This form may occur in somewhat older children.

Symptoms.—**Onset**: Usually sudden, with convulsions and coma, which may last a few hours or a week, and fever. When consciousness returns complete hemiplegia is usually noticed; or it may not become complete until after other convulsions. It is more common on the right side. The **later symptoms** are like hemiplegia in adults. Some cases recover early and quite completely. Less often the paralysis is flaccid. Sensation is usually normal. Aphasia is not rare.

Mental defects may be present—idiocy; imbecility, often increasing with epileptic attacks; or merely weakmindedness. Posthemiplegic movements, such as muscular twitchings, posthemiplegic chorea, or athetosis (see p. 376), are common.

Treatment.—Care in delivery. Cut short convulsions with chloroform and prevent their repetition with bromids. Subsequently, treat the muscles as in adults.

QUESTIONS.

Give the causes and symptoms of cerebral anemia.

Give the causes and varieties of cerebral hemorrhage.

Give the lesions and symptoms of hemorrhage into the internal capsule in all its stages.

What symptoms point to hemorrhage in the meninges, the crus, the pons?

Give the causes and lesions of cerebral embolism and thrombosis, and the points that may help in differential diagnosis.

Differentiate from cerebral hemorrhage and also from one another, coma from the following causes: alcohol, opium, uremia, diabetes, epilepsy, fractured skull.

Give the causes of sinus thrombosis, and its symptoms.

Give the causes, lesions, symptoms, and treatment of infantile hemiplegia.

Describe posthemiplegic chorea. Describe athetosis.

BRAIN TUMORS.

(1) **Tuberculosis** is common in children and young adults and causes the most common brain tumor at this age. It is the large, solitary tubercle (p. 116), and may be single or multiple; it is usually found in the cerebral cortex or cerebellum. It is secondary to tuberculosis elsewhere.

(2) **Syphilis.**—Gumma, often multiple and associated with arteritis, is found in the membranes or on the arteries within the brain. It involves the cerebrum or pons as a rule.

(3) **Glioma,** a neuroglia tumor, blends with the surrounding brain tissue; it is usually soft and very vascular; but may be hard and gray. Microscopically it contains spider cells and fibers, large spindle cells, some of which show hyaline degeneration, cells like ganglion cells. Many of the cells degenerate. It is often very chronic and may be large, or the seat of hemorrhage, or of cyst formation. It is usually in the white matter of the cerebrum, and is single.

(4) **Sarcoma** begins in the meninges of the skull. It may invade the brain or simply press upon it. It is usually at the base and may be round- or spindle-celled.

(5) **Other tumors** are less common; **carcinoma** (metastatic) and almost any other kind of neoplasm occur; the **hypophysis** may be enlarged; **cysts,** usually from encapsulation and absorption of blood or a softened focus, are common; echinococcus or cysticerci may be found.

On the whole, brain tumors are more common in males and between twenty and forty. Tumors irritate or lead to degeneration of brain tissue by (1) pressure; (2) by actual involvement in the tumor; (3) by subsequent inflammation.

Symptoms.—General or Diffuse Symptoms.—(1) *Headache* is present in almost every case; it may be dull and constant, or sharp and paroxysmal; often it is severe, and usually general. Occasionally it may be distinctly localized with local tenderness on percussion of the skull at the same point, and then may rank as a focal symptom. (2) *Optic neuritis* is present in most cases, usually bilateral and intense. It is found by the ophthalmoscope (p. 335); vision may be good for a time, but ultimate blindness is usual. (3) *Vomiting,* without relation to meals and often obstinate, is common with all brain tumors, but especially with those in the pons and cerebellum. These three things occurring without Bright's disease, lead-poisoning or the other causes of optic neuritis are almost diagnostic of brain tumor. There may also be vertigo; psychic disturbances, such as eccentricity, silliness, hysteria, apathy, forgetfulness or perhaps attacks of unconsciousness; convulsions, general or Jacksonian (but this last is a focal symptom), and often *a slow pulse.*

Focal symptoms are given on p. 384, *et seq.* When these symptoms are present they locate the tumor, but the silent regions are many and large.

The *nature of the tumor* may sometimes be inferred from the existence of tuberculosis, syphilis, sarcoma or carcinoma elsewhere.

Prognosis.—Syphilitic tumors usually yield to treatment; other tumors, if not malignant, may become quiescent or grow very slowly, but on the whole the outlook is bad.

Death usually follows in two years, from disease elsewhere, apoplexy from bleeding into the tumor, or exhaustion.

Treatment.—Syphilis and tuberculosis need their treatment. Rarely a tumor may be excised with complete recovery, or some permanent damage.

QUESTIONS.

What are the most important kinds of brain tumors?

Give the general symptoms of brain tumors and tell how to locate the tumor.

What are the prognosis and the treatment?

INSULAR SCLEROSIS.

(Multiple or Disseminated Sclerosis.)

Etiology.—This usually begins between the twentieth and thirty-fifth years or earlier.

Lesions.—The primary change is a hyperplasia of neuroglia in small patches, sometimes as large as 1 in. in diameter, of irregular shape, firm and gray. Microscopically there is an excess of neuroglia cells and fibers. The nerve-fibers are apt to lose the myelin sheath. but the axons are surprisingly resistant, and, therefore, secondary degeneration is not marked. These areas are scattered throughout the brain and cord in any number. They seem more common in the white matter.

Symptoms.—There is a very slow onset which, for a time, may suggest hysteria. The characteristic symptoms develop later. They are (1) **intention-tremor** (volitional or voluntary tremor): (*a*) When at rest the muscles are still, but upon attempted motion they tremble and shake, interfering seriously with manipulations. Thus, water is spilled from a glass before it can be drunk. Sometimes the agitation extends to other muscles than those directly concerned in the movement. (*b*) *Scanning speech* is the result of the tremor of the muscles of articulation, the voice is monotonous, the speech slow, with a pause after every word or even every syllable. Syllables may be elided, *elisive speech*. (*c*) *Nystagmus* results from the tremor of the eye muscles—when the eyes are at rest they are motionless, but when looking at an object there are rapid lateral movements of the eye; or less often vertigo; or rotatory nystagmus.

(2) **Other Symptoms.**—The legs become feeble, have irregular pains, and are stiff. Later in the disease a distinct spastic paraplegia develops, with much exaggerated reflexes and normal electric reaction. Sensory and trophic symptoms are absent. Optic atrophy is rather common. The sphincters are attacked late, if at all. **Mental symptoms**: Apathy and emotionalism are very common, dementia is less common.

Course.—Occasionally there are attacks of coma, perhaps convulsions, with fever, followed by hemiplegia or monoplegia, lasting only a few days. Long remissions or even very marked improvement may take place. A patch of sclerosis may attack any part of the brain or cord, such as a nerve nucleus, or its root-fibers, or the posterior columns, with their corresponding symptoms. After many years the patient dies from respiratory or cardiac paralysis or some intercurrent disease. There is no special treatment.

QUESTION.

Give the chief symptoms of multiple sclerosis.

CHRONIC DIFFUSE MENINGO-ENCEPHALITIS.

(General Paralysis of the Insane, Dementia Paralytica, General Paresis.)

Etiology.—It begins in men from thirty-five to fifty. More than three-fourths have had syphilis. Other factors may be alcohol and the intense modern life.

The **lesions** are chronic productive inflammation of the meninges and arteries, and a chronic progressive degeneration of the neuron. The dura is thick and vascular; the pia is thick, opaque, often adherent to the brain, and edematous. There is an excess of liquid beneath the dura in the pia-arachnoid. The arteries of the meninges and brain are sclerotic. The brain is lacerated in pulling off the pia; it usually weighs less than normal. Convolutions are atrophied and flattened. On section it may be tough and shows patches of hyperemia and anemia. The ventricles are often distended, the ependyma granular. The cord shows similar changes, chiefly in the posterior and the lateral columns, and anterior horns. Microscopically there is new connective tissue in the dura, the pia and around the arteries which also show sclerosis, and collections of leukocytes in the perivascular sheaths. In irregular areas the neurons (cells and fibers) are degenerated, fatty and granular and atrophied. It is supposed to be (1) a primary meningo-encephalitis with secondary degeneration of the neurons; or (2) a primary degeneration of the neuron with secondary sclerosis.

Symptoms.—Prodromal or First Stage.—The symptoms are often trivial but suggestive. There may be irritability, indifference to business, change in the moral character, indecency, sexual depravity, undue fatigue after moderate mental or muscular exertion, and sometimes delusions of grandeur as to wealth, personal prowess, achievements or position. The *onset* is usually very slow and insidious; rarely there may be convulsions or apoplectiform attacks. Motor symptoms (described below) are inconstant, but may be present in this stage, or may even be the first symptoms. This stage may last for years, perhaps with a marked remission or apparent recovery, but in time other symptoms appear.

Second Stage.—*Mental Symptoms.*—Typical mania is usual and the patient is active, restless, sleepless, noisy or often violent; he has delusions of grandeur. Less often there is melancholia or alternate exaltation and depression. *Motor Symptoms:* The face is stolid, the lips and tongue are tremulous; speech is thick and blurred, syllables or whole words being omitted, there is special trouble with certain sounds ("truly rural"); the pupils are often unequal or large, they may show very sluggish reaction, or the Argyll-Robertson pupil. The tremor of the hand and arm interferes with writing, etc. The gait becomes stumbling and either spastic or ataxic. The bladder and rectum may be disturbed. There is liability to attacks of syncope, petit mal, grand mal, Jacksonian epilepsy, or apoplectiform attacks, which may be followed by paralysis for a few days and by a general exacerbation of the disease, or which may kill.

Third Stage.—Finally the patient enters the *stage of paralysis,* and is bedridden, helpless, with relaxed sphincters, loss of motion, of sensation, of mentality; with a liability to bedsores. He vegetates for a variable period, and dies from exhaustion, in coma or convulsions, or of a septic infection or intercurrent disease.

Diagnosis.—This may be made when both mental and motor symptoms are present. The tremor of lips and tongue and the speech are suggestive symptoms.

The patients die in a few years. They should be treated as insane. In syphilitic cases potassium iodid may be given in the early stage.

QUESTION.

Give the etiology of general paralysis, the chief symptoms of every stage, the course, and prognosis.

INFLAMMATION OF THE BRAIN (Encephalitis).

(1) **Acute diffuse encephalitis** is rare: (*a*) It may be hemorrhagic and follow influenza, trauma or alcoholism. (*b*) Or, as an infantile polio-encephalitis, it may attack babies after various infectious diseases. The **symptoms** are acute fever, headache, often convulsions and other head symptoms, coma, and paralysis, usually unilateral. Many die in a few days. Some get well with hemiplegia or other paralysis of the upper-segment type (see also Infantile Hemiplegia, p. 397).

(2) **Abscess of Brain** (*Focal Encephalitis, Cerebral Abscess*).—**Etiology.**—Bacteria: Streptococcus or Staphylococcus pyogenes, pneumococcus, influenza bacillus, and others. The bacteria reach the brain (1) by extension of inflammation from the ear, mastoid, or, less often, from the frontal or other sinuses of the skull, the nose, the orbit, caries of the bones, etc. (2) From trauma—fractured skull or wounds, rarely concussion. (3) In infectious emboli—malignant endocarditis; bronchiectasis, abscess or gangrene of the lung, putrid bronchitis, empyema; liver suppurations; suppurating bone. (4) Also infectious diseases—influenza, sepsis, erysipelas, etc. Cerebral abscess occurs at any age, but is more common from twenty to forty.

Lesions.—The most common sites are the temporal lobe and cerebellum. The large solitary abscess may cause local bulging of the brain with softness. On section there may be reddish pus in a ragged cavity. In older abscesses the pus is encapsulated by fibrous and granulation tissue. The pus in old cases is often sterile; in recent cases it contains bacteria, pus cells, fragments of brain tissue, pigment from the blood, fatty acids, and cholesterin. The pus may be putrid or green or gelatinous. The size of the abscess varies extremely. It may be 2 to 4 in. in diameter. Multiple small abscesses are less common. Surrounding the abscess is a zone of edema.

Symptoms.—There are acute cases with septic fever. In chronic cases fever may be absent or slight. Cases from extension or trauma may show first the symptoms of local or general meningitis. Besides this, the symptoms are those of brain tumor (see p. 398), except that optic neuritis is somewhat less common.

Course.—Even large abscesses may cause no symptoms, or after months the ordinary symptoms may appear. Other abscesses cause very marked symptoms from the start. Acute abscesses may rupture into the meninges or ventricles, often with bleeding and its symptoms. The **diagnosis** is based largely on the existence of the cause of abscess, with symptoms of fever and a brain tumor; local tenderness on percussion and local pain may be distinct. The **prognosis** is bad, but chronic cases may last a long time.

Treatment.—Solitary abscesses that give focal symptoms may be operated upon. Careful treatment of ear disease and suppurations in the head is highly important as a preventive measure.

QUESTIONS.

Give the causes and mode of infection of abscess of the brain.
Detail the symptoms of acute abscess; of chronic abscess.
Give the prognosis and treatment.

HYDROCEPHALUS.

Acute hydrocephalus is tubercular meningitis.

Chronic External Hydrocephalus (*Hydrocephalus ex Vacuo*).—If for any reason the brain is too small for the *cranium*, liquid will fill the space between the dura and the brain. This occurs with congenital smallness of the brain or largeness of the cranium; with the atrophied brain of cachexia, senility, and various brain diseases. The liquid may be diffuse or sacculated. The symptoms are those of the causative condition.

CHRONIC INTERNAL HYDROCEPHALUS.

(1) **Congenital.—Etiology** is unknown, but there may be several cases in one family.

Lesions.—The head is very large and rounded; the face seems to have receded and looks very small; big veins cover the temples; the fontanelles are open; the cranial bones widely separated and very thin. The head may fluctuate. The surface of the brain is flattened, the brain fluctuates like a thin-walled sac. The ventricles and the communications between them are much enlarged, and contain clear liquid (1 qt. in the ordinary case). The brain is a mere shell around the cavities.

Symptoms.—The head may obstruct labor and necessitate craniotomy. Usually a few weeks or months after birth the abnormal growth of the cranium is first noticed, and after a time answers the description given above. There is often a systolic murmur over the fontanelle. The mental development is retarded or there may be idiocy. Epileptic attacks are common. *Motor symptoms:* There is marked muscular weakness, the head wobbles or can not be held up; the child walks very late or not at all. Spastic symptoms are common, with increased reflexes. The *eye*—nystagmus is common; there may be optic neuritis or atrophy or amaurosis. The child usually dies in from one to four years. Rarely they live longer, even to adult life; and sometimes have good intellect.

(2) **Chronic Acquired Internal Hydrocephalus.—Etiology.**—(1) Compression of the veins of Galen or of the iter alone by a tumor; or (2) the closing of the foramen of Magendie and the other communications between the fourth ventricle and the arachnoid spaces by meningitis; (3) there may be no discoverable cause.

Lesions.—In a young child the lesions are like those of the congenital form. After the skull is hard it does not enlarge, but the brain is flattened, smooth, and much thinned; one or more of the ventricles are dilated and full of liquid. There are more or less atrophy and degeneration of the neurons.

Symptoms.—*In a child* it may resemble congenital cases. *In adults* the symptoms are indefinite and the diagnosis is seldom made. There may be headache, vomiting, convulsions, optic neuritis, slow pulse, mental deterioration, epileptic attacks, and spastic paralysis. The **prognosis** is bad, the patients usually dying within a year.

The **treatment** of hydrocephalus is very unsatisfactory. Lumbar puncture and puncture of the ventricles, with or without drainage, are used.

QUESTIONS.

What are the varieties of hydrocephalus?

Give the causes, symptoms, physical signs, and prognosis of congenital and acquired hydrocephalus in a child.

Give the symptoms of acquired hydrocephalus in an adult.

ACUTE DELIRIUM (Bell's Mania).

The **etiology** is unknown. The **lesion** is not distinctive, but there is venous congestion of the meninges and cortex, also some exudation and round cells around the vessels, with petechiæ. There is some degeneration of the ganglion cells.

Symptoms.—*Prodroma* are sometimes present—irritability, restlessness, sleeplessness. (1) **Stage of delirium.**—There are symptoms like acute mania—hallucinations, delusions, incessant, incoherent talking, ceaseless physical activity, jactitation, moving about, and often great violence. After one or two days, from pure physical exhaustion, the patient subsides. Then the (2) **stage of collapse** is said to begin. There may be petechiæ or bullæ; the tongue is dry; the pulse rapid, weak, and very small; the temperature, 102°-104° F.; there are very rapid emaciation, sleeplessness, a low delirium, and, in fact, the typhoid state. The patient dies of exhaustion within a week, or, rarely, not for two or three weeks. Some cases may even get well. The **diagnosis** is made by exclusion of pneumonia, typhoid, acute mania, delirium tremens, uremia, and acute meningitis. Many errors are made. The **prognosis** is not quite hopeless.

Treatment.—Venesection, cold packs or baths, chloral, bromid, hyoscin or ergot; a generous diet of milk, eggs, and beef juice.

QUESTION.

Sketch the symptoms of acute delirium, its prognosis, and treatment.

PARALYSIS AGITANS.

(Parkinson's Disease, Shaking Palsy.)

Etiology.—The disease is rare under forty. It occurs more commonly in those with a neurotic heredity. Perhaps it may follow exposure, worry, mental shock, trauma or possibly an infectious disease. The **lesion** is not known.

Symptoms.—They develop very slowly in almost all cases. (1) **Tremor**: Slow (4 or 5 per second), at first fine, but later very coarse and shaking. It begins usually in one hand, involves the fingers in a pill-rolling movement; the wrist in flexion and extension; the forearm in a rotatory movement. Then another limb is attacked, often a leg, especially at the ankle; finally all limbs and often the face and the tongue; perhaps the head (the eyes also) show a nodding movement. This is increased with emotion or excitement. It stops during voluntary movement, but it affects writing and the finger movements; or it may be stopped by an effort of the will, and is usually absent in sleep. (2) **Weakness** is progressive and in the late stages is very marked. (3) **Rigidity** is important. Movements are slow and often incomplete; passive movements are resisted. (4) The **attitude and gait** result from the rigidity. The head is bowed, the back arched, the elbows held away from the body and partly flexed, the hands often clasped to lessen the tremor, but later the fingers are flexed at the metacarpal joints (the penholding position); the hip and knee are somewhat flexed. Later there may be contractures of the fingers, elbows, or knees. Movements are started and stopped only after a delay; steps are short, stiff, and hurried; at times "festination" or "propulsion" is seen—*i. e.*, the patient seems pushed forward more and more rapidly, and may fall. If pulled backward he may take a few steps and then fall (retropulsion). The face is rigid, blank, and unchangeable, with arched brow, and "mask-like." The lips move slowly in speaking and sentences seem delayed at the start, but are hurried through; the voice may be shrill and monotonous. Sensory and trophic symptoms are absent. Less important symptoms are a sense of internal heat, profuse sweats, restlessness, apoplectiform seizures. The mind is not impaired.

The **course** is slow and progressive; in some stages only one limb or one side may be involved. Toward the end the patient is bedridden. After many years he dies from exhaustion or intercurrent disease. The **prognosis** is bad. **Treatment** is unsuccessful.

QUESTION.

Give the etiology, the principal symptoms, and the course of paralysis agitans.

OTHER TREMORS.

Volitional or intentional tremor occurs during voluntary movement only.

Involuntary tremor occurs when the part is supposed to be at rest.

Simple tremor is a form occurring in certain individuals without known cause. It may last throughout life.

Hereditary tremor is like the above, but runs in families.

Senile tremor is very common in the aged after seventy.

Toxic tremors are due to alcohol, tobacco, lead, mercury, arsenic, opium, etc.

Tremor occurs in hysteria, neurasthenia, epilepsy, exophthalmic goiter, in debilitated conditions, etc.

Tremor occurs temporarily after severe physical exertion, emotion, or fatigue.

QUESTION.

Give the various causes of tremor.

ACUTE CHOREA.

(Sydenham's Chorea, St. Vitus' Dance, Chorea Minor.)

Etiology.—It begins between the fifth year and puberty, or a few years later; is usually in nervous girls; is often a family disease. It often follows emotional strain and the tension of school life, especially at puberty. It sometimes is attributed to endocarditis, reflex irritation (from worms, eye strain, etc.); to certain poisons in anemia, nephritis, or acute infectious diseases. It may occur in pregnancy. Groups of cases are seen. Very often rheumatism is associated with chorea.

Pathology.—Chorea is variously supposed to be an infectious disease, an intoxication or auto-intoxication, a cerebral neurosis or the effect of tiny cerebral emboli. Endocarditis is usually present, probably as effect rather than cause.

Symptoms.—**Motor Symptoms.**—*Choreic movements* are abrupt, purposeless, involuntary contractions of various muscles and groups of muscles. At first slight and hardly noticeable, they soon lead to awkwardness and uncertainty in voluntary action. They often become very complex. They are associated with restlessness that may become extreme. The hand is first affected, as shown by awkwardness in writing, sewing, and eating. The face also twitches, the tongue when protruded jumps about, speech may be difficult or even unintelligible, the head may jerk. The legs and trunk are affected later and to a less degree, but the gait is often shuffling or sidling or jumping. The trunk muscles may cause irregular respiratory movements. At first but few muscles are involved; later, large groups. It may be confined chiefly to one arm, or both, or to one side of the body (hemichorea). The force and the frequency of the movements may be slight or extreme. The movements are usually absent in sleep, and are increased by attention, emotion or fatigue. Some muscular weakness may appear, but it is seldom marked. The sense of fatigue usually is slight. **Mental Symptoms.**—The patient is peevish, irritable, emotional, lacrimose and shows mental restlessness. Rarely symptoms like mania may develop (chorea insaniens), especially in pregnancy. The heart often becomes rapid and palpitates, and functional murmurs are very common. Endocarditis, acute or chronic, is found in perhaps 50 per cent. of the cases; usually it is mitral regurgitation. **Sensory** and **trophic symptoms** are not important, but herpes may appear. Rheumatism may precede, accompany or follow. Fever is absent or transient.

Course.—The onset is slow. In mild cases the choreic movements and psychic disturbances are slight, and recovery follows in from two to four weeks. In the severer cases the movements are pronounced, and the patients may be unable to study, to work, or even to walk or to feed themselves. They are apt to show much irritability, loss of power of attention—perhaps distinct mental dulness—anemia, and loss of weight and strength. The disease lasts from two to four months or longer. The worst cases have maniacal delirium and often are fatal.

Prognosis.—Perhaps 2 per cent. die; very few become chronic and last many years; most recover slowly, but are very apt to relapse, especially in spring or after fright or other emotion.

Treatment.—The application of reasonable hygiene in school and home, especially for nervous girls, will prevent many cases and relapses. Even mild cases should be kept from school, and ordinarily in bed, shielded from all excitement and well fed. Slight digestive disturbance and constipation need prompt attention. **Drugs.**—Arsenic is gradually increased from 2 drops of Fowler's solution, three times a day, to the limit of toleration. Cimicifuga, strychnin, and iron are used. Bad cases may need hypnotics and sedatives, such as chloral and bromid, and also complete seclusion from friends. With paresis or great weakness in convalescence, massage and electricity are used. At times chorea in pregnancy may demand abortion.

QUESTIONS.

Give the causes, symptoms, prognosis, and treatment of chorea minor.

Discuss the relation of chorea, heart disease, and rheumatism to one another.

OTHER CHOREIFORM DISORDERS.

Habit spasm (habit chorea) occurs usually in children from seven to fourteen, especially girls, with neurotic temperament and heredity and in poor general health. There is an habitual muscular contraction—such as a grimace or sniffing, shaking the head, shrugging the shoulder, or other more or less odd movements of head or arms. It lasts for a few months and usually disappears, but sometimes it persists indefinitely. The treatment is to improve the general health in every way, to remove excitement, and to give arsenic as in chorea.

General or **impulsive tic** (Gilles de la Tourette's disease) is a psychosis, often suggesting monomania. It usually occurs in persons with strong neuropathic heredity, and begins in childhood and after emotional states. **Symptoms:** (1) The repetition of winking, grimaces, or more complex but well-coördinated movements of the hands or even of all limbs. These are hardly subject to voluntary control. (2) With the movement, or at other times also, there may be the uttering of some senseless sound, such as barking or crowing; or a profane or obscene expression (coprolalia); rapid repetition or mimicry of words (echolalia); or the imitation of actions of others (echokinesis). (3) Sometimes they have an obsession or fixed idea about objects, words or mental processes (onomatomania, arithmomania, etc.). Occasionally recovery follows. Usually the disease persists, but does not shorten life. General hygienic and tonic treatment is indicated.

Saltatory spasm (latah, jumpers) is a disease of neuropaths, especially adult men. There are spasms of the calves, causing jumping movements which may lift the patient from the ground, so that he falls. The spasm occurs only when the foot is pressed upon or the patient stands. The disease may last any time, from a few days to years, but recovery usually follows.

Chorea major (pandemic chorea) is the epidemic jumping, dancing, and gesticulation associated with intense religious excitement.

Reflex or **hysteric chorea**, rhythmic movements of special muscles, such as salaam or head movements, occurs in hysteric persons.

Electric chorea is a term applied to some endemic and other rare spasmodic conditions.

Paramyoclonus multiplex (myoclonia) occurs in females after emotion, fright, or muscular strain. It is sometimes hereditary and is seen in neuropaths. There are rapid clonic contractions of groups of muscles, roughly symmetric, in the head, shoulders or thigh. The reflexes are exaggerated, the electric reactions normal, sometimes there are tender points over the spine. Some patients recover after a long time. It does not kill. **Treatment**: Galvanize the tender points over the spine, give general tonic treatment and arsenic and strychnin.

Chronic Chorea.—(1) **Hereditary adult chorea** (Huntington's chorea) is strictly hereditary through several generations, beginning from thirty and fifty, usually occurring in neuropaths. The *lesion* is probably a chronic meningo-encephalitis, with atrophy of the cortex. *Symptoms:* A gradual onset of irregular movements of the face and one or more limbs; incoördination interfering with writing, sewing, etc., and causing an unsteady gait, and blurred pronunciation; mental impairment, irritability, and finally dementia. It lasts many years and seems incurable.

(2) **Senile chorea** develops sporadically, usually after fifty-five. It is like the above, without the mental symptoms. Some cases recover.

Prehemiplegic chorea may occur in limbs about to be paralyzed, during the two or three days immediately preceding an apoplectic attack.

Posthemiplegic chorea may follow infantile hemiplegia (rarely other hemiplegia) associated with hemi-anesthesia. It is due to destructive lesion of the posterior parts of the internal capsule and optic thalamus. The movements are rather slow, are associated with athetosis, and last throughout life.

Athetosis (see p. 376).—(*a*) **Symptomatic athetosis** occurs in most cases of infantile hemiplegia on the affected side, also sometimes in idiocy, epilepsy, spinal paralysis of children, and possibly tabes; very rarely after adult hemiplegia. (*b*) **Idiopathic athetosis** occurs in hereditary neuropaths without discoverable cause. Also, it may possibly be due to difficult labor, especially if instrumental. Except for the athetosis the muscles are normal. The movements occur in the head, neck, and upper extremity. The *lesion* is unknown. The disease may begin in adult life and lasts until death.

QUESTION.

Describe habit spasm, general tic, hereditary chorea, posthemiplegic chorea, and athetosis.

INFANTILE CONVULSIONS (Eclampsia of Infants).

Etiology.—Eclampsia is a sudden attack of general convulsions. Infants are very liable to convulsions: (1) **Representing the chill** of later years at the onset of acute fevers, especially pneumonia, scarlatina, measles. (2) **Reflex causes:** (*a*) Possibly teething; (*b*) an overloaded stomach with indigestion; (*c*) intestinal parasites (round worms); (*d*) phimosis or adherent prepuce; (*e*) otitis. (3) **Lesions of the brain:** Hemorrhage at birth or later (infantile hemiplegia), acute encephalitis, acute poliomyelitis, tumors, hydrocephalus, gliosis, etc. (4) **Meningitis** from any cause. (5) With **debility,** especially from protracted diarrhea. (6) Possibly from fright, or congestion of the brain. (7) **Uremia.** (8) Possibly an **auto-intoxication.** (9) In those who later develop epilepsy. (10) Without discoverable cause. (11) Finally and most important, **rickets,** with or without some exciting cause, reflex or other, is present in about one-third of the cases.

Symptoms.—Marked cases have rather typical epileptiform convulsions, with rolling eyes, twitching, tonic and clonic spasms, etc. These may recur frequently for one or several days without return of consciousness (status eclampticus). The spasms may be Jacksonian in type. The mild cases may show only fixation of the eye, a dusky pallor, and perhaps slight twitching that lasts but a few seconds. Laryngismus stridulus is analogous.

There may be but a single fit or many, repeated at varying intervals, even for days or weeks. Sometimes the convulsion is fatal, especially in a weak child, or it may induce cerebral hemorrhage. The causative condition must always be considered in the **prognosis.**

Treatment.—The fit: chloroform inhalation, amyl nitrite, hot baths with cold to the head, or cold alone, and perhaps morphin hypodermically. Later, chloral, bromids or antipyrin. The cause must be discovered, if possible, and treated. If no cause can be found and the convulsions recur, bromids should be given.

QUESTION.

Give the causes of convulsions in children, their symptoms, and treatment.

EPILEPSY (Falling Sickness, Falling Evil, Morbus Sacer).

Etiology.—(*a*) **Heredity.**—The parents' family shows a neuropathic taint, usually insanity, sometimes epilepsy, chronic alcoholism is common, and perhaps hysteria may predispose. (*b*) **Age**: About one-fourth of the cases have the first attack in the first decade; half of them in the second; and most of the remainder in the third. It appears earlier with marked heredity. (*c*) While a general tendency to nervous instability is the chief factor, the disease may follow, after a longer or shorter interval, the following conditions: (1) Infantile convulsions (probably due to rickets); (2) meningeal hemorrhage or other cortical lesion with or without paralysis (these are common causes in children, but rare in adults); (3) trauma, such as a blow on the head, or cerebral concussion; (4) toxic conditions, such as scarlet fever, possibly nephritis, intestinal auto-intoxication, a prolonged alcoholic excess, lead-poisoning, etc.; (5) intense fright, perhaps prolonged anxiety; (6) reflex causes—intestinal worms, possibly the irritation of a nerve in a scar, trauma anywhere, polyps or foreign body in the nose or ear, phimosis, biliary or other colic; (7) heart disease (Stokes-Adams syndrome); (8) pregnancy—sometimes attacks start in pregnancy, in other cases the attacks are absent during pregnancy and perhaps during lactation also.

Idiopathic epilepsy has no discoverable lesion. **Symptomatic** or **organic epilepsy** is associated with a lesion in the brain. The line between them can not be sharply drawn, for while at times the epilepsy stops when this exciting cause is removed, there are other cases in which the attacks persist, the lesion having simply started them. Symptomatic epilepsy occurs with tumors, abscesses, hemorrhage, local trauma, and especially syphilis of the brain. It is usually of the Jacksonian type, but generalized attacks may occur. The attack is supposed to be a sudden explosive liberation of nervous energy, wholly involuntary.

Symptoms.—(1) **Major Epilepsy** (Grand mal).—*The Attack.*—An *aura* often is present for a few seconds. It may be *sensory*—numbness, tingling, etc., at one point, especially epigastric uneasiness, or it may be more general; *motor*—contractions or perhaps paralysis of a certain muscle; *vasomotor*—pallor; or *sensorial*—a dreamy state, or terror or a subjective sight, sound, taste or smell. A given patient has the same aura before all attacks. Rarely, unconscious acts described below may be seen. Then the patient utters a *loud cry* and falls in coma, often injuring himself by the fall. He becomes pale and rigid; has a general *tonic spasm* of the entire body and soon grows dusky. After a few seconds, *clonic spasm* starts and quickly becomes very marked, involving the entire body; the jaw chews and grinds; the tongue is agitated and very often bitten; frothy and bloody saliva is forced out of the mouth; the limbs and trunk shake violently; respiration is irregular, intermittent, and noisy; the jugulars stand out and the skin grows cyanotic; urine and semen are often, and feces sometimes, discharged; pupil reflexes are absent. The convulsion subsides in from one to five minutes.

After the attack, usually, the very deep coma gradually changes to a deep relaxed sleep, lasting one hour or several; or the patient may awake at once; the mental state is often confused; usually the muscles ache, the patient may be irritable for a few days, or have a headache, or at times may feel unusually well. Before the return of consciousness he may have "postepileptic states," marked by *automatic movements,* such as turning over into the prone position, undressing or moving about, or he may be maniacal and violent, even murderous. Later he is unconscious of what he did. Rarely, paralysis of one or more limbs may appear and last a few days, or there may be sensory aphasia. Just after the fit the deep reflexes are absent. Later they are exaggerated for a time.

During the fit there may be bleeding into the skin (petechiæ of the face and neck, hematoma of the external ear); or into the mucous membranes or even into the cranial cavity.

The Sequence of Attacks.—The attacks may be very infrequent; or recur at somewhat regular intervals, often two or three weeks; or they may occur in groups with long intervals between. The *status epilepticus* is a succession of attacks every few minutes, without return of consciousness, with fever, often hyperpyrexia, and death. Attacks may be diurnal or nocturnal. Nocturnal attacks may be diagnosed only by finding in the morning a bitten tongue, blood-stains on the pillow, evidence of urination, headache, mental confusion, aching muscles, petechiæ, etc.; or the patient may roll over, bury his face in the pillow, and die of asphyxia. After a number of attacks there is usually distinct mental deterioration, but some men of genius have had epilepsy.

(2) **Petit Mal** (*Minor epilepsy*).—There are no convulsions, but, with or without an aura, attacks of giddiness, faintness, and pallor, followed by unconsciousness for a few seconds. There may be such automatic unconscious acts as were described above (masked epilepsy). Perhaps there is some jerking of the limbs or tremor. Attacks of petit mal recur at varying intervals. Usually major attacks come on also, and the minor attacks may alternate irregularly with them. Sometimes, however, the minor attacks occur alone.

(3) **Jacksonian, Cortical, Symptomatic, or Partial Epilepsy.**—Usually there is an irritative lesion in the motor area, possibly in the sensory area of the cortex. Coma is late or wholly absent. There may be numbness or tingling of the thumb, foot, face or any other part, followed by twitching. This spreads to the successively contiguous centers in the motor area. The convulsions may be limited to muscles represented in a smaller or larger area of the cortex, such as one limb or one side of the body; or they may become general. In time these patients are apt to have general convulsions, and perhaps typical major attacks.

Prognosis.—Death in a fit is rare, but the patient may fall into a fire or water, or he may be asphyxiated by a foreign body in the larynx, or by his pillow. The status epilepticus is often fatal. Jacksonian or reflex cases may get well if the source of irritation is removed. Occasionally other cases seem to stop. If attacks are infrequent enough the mind may not be affected. Syphilis of the brain, tumors, etc., may be curable, but on the whole the prognosis is very bad.

The **diagnosis** must be made from hystero-epilepsy, uremia, and other forms of coma. Recurring attacks of convulsions in adults over thirty who were previously well and have good heredity are usually (90 per cent.) due to organic brain disease. Jacksonian epilepsy may be perfectly simulated in uremia, general paresis, with tumors, clots, etc.

Treatment.—The attack may occasionally be aborted by blistering the site of the aura, or ligating the limb above it, or by amyl nitrite. During the attack a cork or rolled handkerchief should be put between the patient's teeth, to save the tongue. The clothes should be loosened and he should then be let alone. In long attacks or the status epilepticus, chloroform or amyl nitrite inhalation, or morphin hypodermically may help. To lessen the recurrences the patient should have physical work and freedom from excitement, general hygienic care, good food, cleanliness. The moral stamina should be carefully developed in children to counteract, if possible, their neuropathic tendencies. When the mind deteriorates or violence appears the patient should be confined in an asylum. Epileptics should not marry. The diet should contain little meat, and should consist of light food in small quantities, the bowels should receive careful attention. **Drugs**: Bromid of sodium or potassium, about ʒj a day, is given for at least two years after the latest attack. Arsenic may lessen the chance of bromism. In petit mal, nitroglycerin, to its physiologic effect, may help. Surgery is clearly indicated in Jacksonian epilepsy. The fits may cease for a few months or even permanently after any surgical operation anywhere in the body.

QUESTIONS.

Give the etiology of epilepsy.
What are the varieties and symptoms of each type?
What are postepileptic states?
Give the prognosis and treatment.

TETANY.

Etiology.—This is not rare in the first three years with rickets, and in the second and third decades. It is more common in young boys and adult women. Usually there is some definite preceding condition, such as rickets, dilation of the stomach (especially after lavage), severe diarrhea, worms, pregnancy, lactation or acute infectious disease (pneumonia, cholera, measles or small-pox). It may follow thyroidectomy. It probably is often an intoxication or auto-intoxication. It occurs in epidemics in certain parts of Europe.

There is no known **lesion.**

Symptoms.—The Attack.—There is a *tonic spasm*, usually bilateral, in the hands and often in other parts of the body also. The fingers are straight, drawn together, and flexed on the metacarpus; the thumb is opposed and often flexed. Sometimes the elbow may be flexed, the arm adducted in front of the body. The toes are drawn together and flexed, the ankle extended and inverted, the knee straight. The face and trunk may be involved. Laryngismus stridulus is sometimes attributed to tetany. *Sensory symptoms:* Paresthesia may precede the attack. The rigid, hard muscles are painful and tender. Hysteric stigmata may be present. *Vasomotor and trophic symptoms:* The skin may be tense and look edematous; herpes may appear. The spasm may last for a few minutes or a few days. Relaxation is only partial during sleep. There may be a single spasm; or there may be several a day, in which case some fever is usually present. In *acute cases* the spasms cease after a few days. In *chronic cases* they recur for weeks or months; this type is more common in adults.

Between Spasms.—The motor nerves to the involved muscles are oversensitive to mechanical and electric stimuli (Erb's symptom). Slight stimuli over the sensory nerves of the limb cause paresthesia (Hoffman's symptom). Pressure for a few moments on the large vessels or nerves of the limb induce the spasm (Trousseau's symptom). Pressing upon or tapping the facial nerve causes a spasm of the face (Chvostek's symptom). These persist as long as the spasms are apt o recur.

Prognosis.—The patients recover, but may have relapses again and again for years.

Treatment.—In the attack use bromids, cold baths, chloroform inhalation. **Causal treatment** is most important. Avoid stimulants and indigestible foods. Use general tonic treatment.

QUESTION.

Give the etiology, symptoms, prognosis, and treatment of tetany.

OCCUPATION NEUROSES (Professional Spasm).

This occurs in penmen, pianists, violinists, telegraphers, typewriters, milkmaids, cigarette rollers, seamstresses, weavers, ballet dancers, motormen, and many others.

Writers' cramp (*scriveners' palsy*) is seen usually in adult males who write much and in a cramped fashion, and are neurotic. There is no known lesion.

Symptoms.—The neurotic symptoms are often exaggerated for a time, and then the trouble in writing slowly develops. There are various types: (1) *Spastic type:* This is the most common. When writing, especially for a long time, painful spasms develop in the flexors or extensors of the thumb, fore-, and middle fingers. The writing gets worse and worse until it is illegible, or writing may be impossible. There may be local paresthesia. In extreme cases the wrist, forearm, and even upper arm may be affected. Mental distress increases, there is much anxiety, occasionally profuse sweating, and palpitation. There is tenderness over the muscles, often tender points along the nerves involved, and sometimes over the thoracic spine. Changing the pen or the penholder, or the position, or even learning to write with the other hand, may relieve—but usually only temporarily. (2) *Tremulous type:* There is tremor upon attempting to write. (3) *Neuralgic type:* There are severe pains in the muscles, without spasm, when attempting to write. (4) *Paralytic type:* In writing, fatigue develops rapidly and, finally, the hand becomes powerless to write.

All other movements of the muscles are quite normal. The condition is obstinate and often incurable, but may improve greatly for a time. It must be diagnosed from other conditions interfering with writing, such as chorea, paralys agitans, progressive muscular atrophy, tabes or hysteria.

Treatment.—Stop writing altogether for weeks or month Galvanism may be applied over the tender points and th affected muscles and their nerves. Treat the general nervou state.

QUESTION.

Give the etiology, symptoms, and treatment of writers' cramp.

MIGRAINE (Megrim, Hemicrania, Sick Headache).

Etiology.—This is common in neurotic, gouty, and rheumatic families; more common in women, especially at menstruation. It usually begins in early childhood or puberty and but rarely after twenty-five. No exciting cause for a attack may be discoverable, or it may follow reflex causes suc as eye-strain, polyps or foreign bodies in the nose, adenoids, carious teeth, indigestion, emotion, fatigue or worry, uterin or menstrual disorders. At times an attack has replaced o alternated with epileptic attacks. Theories as to its causation: (1) It is a neurosis of the cervical sympathetic, an attack being induced and accompanied by overaction of the cervical sympathetic on the affected side. As the attack wanes there may be sympathetic paralysis. Others say there are two types, one described above due to sympathetic overaction, the other due to sympathetic paralysis on the affected side. (2) It is a sudden discharge of nervous energy affecting certain sensory nerves, and is therefore the sensory equivalent of epilepsy. (3) It is due to diminished excretion of uric acid.

Symptoms.—*Prodroma* are often present: depression, languor, nausea, vertigo, or such subjective sensations as scotomata, flashes of light, zigzag lines (fortification spectra) often colored brilliantly, visions of animals, hemianopia; rhythmic movements of the pupil on affected side. Less often other special senses are similarly affected, or there may be paresthesia, pain or muscular cramp on the affected side, or, rarely, sensory aphasia. These are, after a while, replaced by *headache*, often beginning at a small spot on one temple or one side of the forehead and spreading over all that half of the head. It is very intense, and may extend to the neck or arm; it is increased by movements or a strong light or loud sound. Not rarely it is bilateral. Often there are anorexia, much nausea, and vomiting of undigested food. Vomiting may mark the end of the attack. **Sympathetic nerve symptoms**: (1) Spastic type—the affected part of the head is pale and cool; the arteries small and hard, and the pupil of that side dilated. Later, there may be symptoms of sympathetic paralysis. (2) Paralytic type—that side of the head is flushed, the arteries large and pulsating, the pupil contracted. Later, there may be symptoms of a spastic condition. A person may always have attacks of one kind, or both kinds may alternate, or there may be no consistent sympathetic symptoms. After a few hours or, perhaps, not for a day or two, the symptoms abate, the patient sleeps and awakens free from pain, but exhausted. Attacks may recur with remarkable regularity, or be irregular. They are common toward the end of menstruation. They are apt to cease after middle life. The general health and mental power are unaffected. During and before the attack the output of uric acid may be lessened.

Treatment.—Avoid the cause of an attack, which must be carefully sought in every case. The drugs used are nitroglycerin, given continuously for a long time; strychnin, arsenic, iron, and cannabis indica. Some attacks may be aborted or relieved by lavage; or sodium salicylate, ʒj; or a cathartic; or hot, strong coffee; or chloroform, gtt. xx; or vasodilators or vasoconstrictors; or phenacetin, etc. The patient should stay in bed in a dark, quiet room, with no food. Treatment is very often disappointing.

QUESTIONS.

Give the etiology, symptoms, and treatment of migraine.
Discuss the relation of the sympathetic nerves to migraine.

NEURALGIA.

Etiology.—It is more common in neurotic families; in females; in adults; with debility; following anemia, malarial or other cachexia, lactation, menorrhagia, overwork, or insufficient sleep; with various gastro-intestinal or genito-urinary disturbances; in the rheumatic or gouty, lead-poisoning, diabetes, nephritis, perhaps in typhoid or malaria, often in syphilis. The attack may be induced by exposure to cold, local irritation; or reflexly, as from a carious tooth, irritation of a nerve in a scar, a neuroma, pressure from a tumor or aneurysm, caries of bone, trauma, or from the female pelvic organs. Long nerves and those exposed to pressure in bony canals are most vulnerable.

There is often no discoverable **lesion**; at other times a neuritis is found.

Symptoms.—**Paroxysmal pain** along the course and distribution of a nerve, usually unilateral, aching, boring, or stabbing in character. It may last a few seconds and recur every few minutes; or last for minutes or hours and recur at various intervals. **Tender points** are present, usually where a nerve lies on a bone or gives off a superficial branch. There may at first be hyperesthesia, later anesthesia or numbness. **Vasomotor** and **trophic** symptoms may be present—pallor or flushing with throbbing of the arteries, rarely some edema; local sweating. The hair may turn gray or fall out; or herpes or erythema occasionally appears in the affected region. Sometimes there is local muscular twitching. All these symptoms are usually limited to the distribution of the affected nerve. Periodicity is sometimes striking with no association with malaria, the attack recurring every day at the same hour, or at longer uniform intervals.

Special forms of neuralgia should be carefully studied with the help of charts showing the distribution of sensory nerves.

(1) *Trigeminal neuralgia* (tic douloureux) is very common, often severe, and may involve one, two or, rarely, all divisions of the nerve. *Tender points:* (1) Ophthalmic division—at the supra-orbital notch, sometimes the inner angle of the orbit, over the upper eyelid or the eyeball. (2) Superior maxillary division—at the infra-orbital foramen; and in or above some of the upper teeth. (3) Inferior maxillary division—at the mental foramen, over the auriculotemporal branch above the zygoma, and at the tip of the tongue.

(2) *Cervico-occipital neuralgia* (I–IV C.) is often confined

to the posterior branches. A tender point is present just below the mastoid process. The scalp may be tender.

(3) *Cervicobrachial* (IV C.–I T.) *neuralgia* is often limited to the circumflex or the ulnar nerve. Tender points—over the ulnar nerve at the elbow, over the musculospiral nerve three inches above the outer condyle.

(4) The *phrenic nerve* is rarely attacked. The pain is intensified by movement of the diaphragm. Tender points are found at the insertions of the diaphragm at the free border of the ribs.

(5) *Intercostal neuralgia* (I–XII T.) is very important, usually involving the middle intercostals and on the left side. It includes the inframammary pain (mastodynia) common in nervous hysterical women. The pain is often severe; it is continuous or paroxysmal. Herpes is rather common. Tender points—(1) dorsal, two or three inches from the spine; (2) in the midaxillary line; and (3) sternal—just lateral to the sternum; (4) sometimes in the breast tender lumps are felt.

(6) *Lumbar or Lumbo-abdominal Neuralgia.*—The pain is usually over the iliac crest, in the inguinal canal, spermatic cord, scrotum or labium majus. There are tender points at the above places and also in line with the tender points of the intercostal nerves.

(7) *Coccygodynia*, usually in women, is often persistent. The pain is worse in the sitting position or with straining.

(8) *Great sciatic neuralgia* (see p. 353).

(9) *Painful heel, plantar neuralgia*, and *metatarsalgia* (fourth metatarsophalangeal joint) are described.

(10) *Visceral neuralgias* occur in various viscera and include many cardiac, gastric, and intestinal neuroses, being especially common in the ovaries. Many of them have been described under these various headings.

(11) *Reflex neuralgias* are not rare. Irritation at some point causes pain, perhaps at a distance, often along another branch of the same spinal or cranial nerve. Deep viscera are often the seat of irritation, and the pain may be referred to cutaneous or other areas. Examples are the knee pain in hip-joint disease, the right shoulder pain in liver abscess, the pain in the thigh with renal colic. Head's table (p. 357) will help interpret many of them.

Treatment of Neuralgia.—The **local cause** should b removed if possible, such as a bad tooth or a painful scar **General causes,** such as debility, anemia, gout, and the others need treatment. The removal of the cause is the most successful part of the treatment. The attack: *Locally*—heat o cold; the ascending current or a mild faradic current ove tender points; blisters, or other counterirritants; acupuncture the injection of distilled water; and all sorts of anodynes liniments, and ointments are used. *Drugs*—phenacetin an similar drugs, aconite, cannabis indica, gelsemium, arsenic quinin, croton chloral, strychnin, and very many others ar used. Morphin and alcohol are very efficient, but are particularly dangerous. Nerve-stretching, neurectomy, or excision of certain ganglia may help.

QUESTIONS.

Give the etiology, general symptoms, and treatment of neuralgia.
Give the tender points and the distribution of the principal varieties
What is reflex neuralgia?
Give some forms of visceral neuralgias.

HERPES ZOSTER (Zona, Shingles).

Etiology.—A common disease; it occurs at any age, but usually under twenty-five. Probably it is a specific disease, and perhaps infectious; at any rate, it is epidemic at times It occurs secondarily also in many diseases of the central nervous system. No exciting cause can be found, as a rule.

The **lesion** is an acute inflammation, often hemorrhagic, of one or several posterior spinal ganglia on one side of the body, with degeneration of some ganglion cells and consequent secondary degeneration in the nerve and cord. There are also the skin lesions and large tender lymph nodes.

Symptoms.—The onset may be acute with a chill and fever in primary cases and pain in the distribution of the nerves from the affected ganglia. In three or four days the rash appears, usually strictly limited to the distribution of the affected nerve or nerves—the skin is reddened, then groups of vesicles appear, with contents at first clear, later turbid: they may dry up, or break and form crusts; they may be rather deep and leave ulcers and scars. The average duration is a week or two. The most troublesome symptom is **neuralgic pain,** often severe, especially in the aged, which may last many weeks or months. Intercostal, or lumbar nerves, and the supra-orbital or other divisions of the fifth nerve are the most common sites, but any nerve may be involved. It is practically always unilateral. The particular ganglion or ganglia involved may be found by comparing the site of the rash with charts of the cutaneous nerves. Mucous membranes are often affected. Second attacks do not occur in the same nerve roots, but they may occur elsewhere.

Treatment.—Locally: protective applications, such as zinc oxid powder or collodion. The pain is treated as in neuralgia.

QUESTION.

Give the lesions and symptoms of zoster.

HYSTERIA.

Etiology.—Hysteria is a psychosis, with defect of controlling power in the highest psychic centers and a lack of emotional equilibrium. A **predisposition** exists and is very marked in neuropathic families (those showing insanity, epilepsy, hysteria, perhaps neurasthenia also). This **tendency is fostered** by (1) a training that fails to teach rigid self-control, is indulgent, sensuous, luxurious, selfish; (2) a life that is too stimulating or demands too much nervous energy in school, business or society; (3) by morbid contemplation of self; (4) by general deterioration of health from any cause, such as a bad mode of life, too hard mental work or worry, physical exhaustion or a protracted weakening disease, alcoholism or morphinism, etc. An **attack may be induced** by an emotional state, either intense and sudden or more protracted, such as terror, grief, great joy, diappointment, shame, anxiety, worry. This emotional state may have been excited by physical causes really important or so trivial that a normal person would hardly notice them, such as an unexpected sight or sound, pain or mere discomfort in any part of the body from any cause, perhaps trauma, often nervous disease. In some cases this clear sequence can not be made out. Hysteria may be a contagious imitation of other hysterics. excess and masturbation in both sexes are given as ca

Hysteria is very common in women, but not rare i It usually begins at or a few years after puberty, often third decade, occasionally at earlier or later periods. much more common in certain nationalities, especia Jews and the French.

Symptoms.—Mental Symptoms.—The patient is ir emotional, may laugh when she should cry, etc. Inste customary emotion she may have an hysteric fit. Sh the power of concentration and attention, is cunning ceptive, courts sympathy, is introspective. There may lucinations or delirium, or even real insanity. O attacks there is unconsciousness, real or only appare later, the patient may show that she heard and saw p what was going on. *Trance* or *lethargy* may occur sp ously or follow a convulsive attack. There are pallor, heart action, real or seeming unconsciousness. Such have been buried alive. Practically this is spontaneo nosis and it may last any length of time, even months. teric persons are favorable subjects for hypnosis, and r hypnosis greatly aggravates the hysteria.

Sensory Symptoms.—Hyperesthesia and spontaneo occur in irregular areas, but the hyperesthesia disapp the patient's attention be directed to some other spot. times pressure on such an area induces an hysteric such spots are called *hysterogenic zones.* They are ofte in the ovarian region, sometimes in the thorax, breast o of the vertebræ. There are attacks of violent pain in viscera, like that of real disease. Neuralgias may Paresthesias are common. Areas of anesthesia and an small or large and irregular, are found. There often i anesthesia, usually on the left side, but the loss of se may have any other distribution, and may shift fr place to another. The patient may be pricked with without pain. Tickling the pharynx and palate ca nausea; the reflex closing of the eyelids following c tival irritation is absent. The anesthesia often invol deeper structures also, and there may be loss of m sense. The patient is not aware of this sensory pa

Special senses: Sight is usually affected. There may be dimness of vision, restriction of the visual field, especially for certain colors (achromatopsia), amblyopia, or occasionally amaurosis. *Hearing* may be lost or feeble or very acute in one or both ears. *Smell* and *taste* may be lost or there may be subjective smells or tastes, often very distressing. With hemianesthesia the special senses are often involved.

Motor Symptoms.—Tremors are common, especially in the eyelids and fingers. In the limbs there may be a voluntary, an involuntary, or a constant tremor. Local or general twitchings, or general convulsions, clonic or tonic, occur. Spasm of the pharynx and esophagus causes the "globus hystericus," a feeling as if a ball were rising in the throat; there may be spasm of the glottis, peculiar sounds, etc. There may be hiccough. Spasm of the muscles of the abdominal viscera may cause phantom tumors, such as spurious pregnancy (pseudocyesis). These tumors may last years, but they vary in size. Contractures of the limbs may develop. Other motor symptoms are *catalepsy* (a limb stays in any position in which it is placed—this may be present only when the eyes are closed) and various *choreiform* disorders. It is very important to remember that all these muscular symptoms disappear in anesthesia and often in sleep. *Paralysis:* Mono-, hemi-, or paraplegia is often associated with anesthesia in the same area. These may be spastic or flaccid, but the muscles do not waste, nor are the electric reactions changed. The reflexes vary, but are often increased. Paralyses are most marked in the legs, and are usually slight or absent in the face. Aphonia is common and there may be mutism.

Vasomotor, Trophic, and Secretory Symptoms.—There may be local pallor, flushing and sweating, salivation, increase or decrease of the secretions of the stomach and intestines, sometimes a mucous diarrhea or membranous colitis. Polyuria is very common; a great decrease in the amount of urine is less common. Real trophic disorders are very rare, but swellings may occur in a limb or smaller region.

Self-mutilation (probably always in anesthetic areas), asthmatic attacks, very rapid breathing, palpitation, tympanites, peristaltic unrest, spitting or vomiting of blood (probably from the mouth and pharynx), and many other symptoms are common. There may be petechiæ in the skin. Fe rare, but even hyperpyrexia may occur.

Convulsive Hysteria.—(1) **Minor forms** are co They are marked by acute attacks of various symptom the above list, and often follow a sudden emotion. Fai twitchings, convulsions, and tympanites are common. are followed by polyuria and mental depression.

(2) **Major Forms.**—(*a*) Prodromal symptoms—the nary hysteric manifestations, especially at hysterogeni and the globus hystericus, may last a few minutes. there follows (1) the *epileptoid condition* (hystero-epil This differs from epilepsy in the following points: It u follows emotion; the warning symptoms are ordinary h symptoms, such as palpitation, malaise or choking; the is often gradual; they lie down rather than fall; often s throughout the fit; there is rigidity, often opisthotono prosthotonos or pleurosthotonos; the active movemen more purposeful; they struggle, strike themselves and persons; they writhe, grasp objects, etc.; they bite thei their hands or other persons, but not the tongue; th no incontinence; the fit lasts several minutes (ten or it may be stopped by ice, water, etc. Fits may be re frequently without exhaustion or anything suggesti status epilepticus; the patient will show hysteric sti (2) This may be followed by the *period of clownism* emotional display, odd and very difficult gestures and tions. The religious jumping and dancing, beast mimic spurious hydrophobia are of this class. (3) The *em* *or dramatic* stage may follow. The patient assumes dr poses that seem the direct result of hallucinations and sions, and illustrate emotional states, such as fear, joy, e religious devotion. There may be catalepsy or trance. The final stage is that of *delirium* of a somewhat mil usually with depression and a return of consciousness. is no muscular activity, but often there are paralyses, n or hallucinations which may last for weeks. In typical attacks all these stages are present, but in this countr are very seldom seen. The epileptoid stage, however, i mon without the others.

Hysteric Stigmata.—Some stigmata are always between the convulsive attacks and should be looked for in every case of suspected hysteria. Some of the more important and common are loss of conjunctival reflex, anesthesia of the pharynx, areas of cutaneous anesthesia and analgesia (especially hemianesthesia), limitation of the visual field, achromatopsia, changes in the sense of hearing or taste or smell, quivering eyelids, etc.

The Course.—The **milder cases** show only a general lack of emotional balance, a tendency to work for sympathy and to exaggerate all physical and mental troubles. **Severer cases** often have sensory and motor paralysis, convulsions, contractures, and other such symptoms. Longer or shorter remissions occur, but relapses are very common, because the emotional nature of the patient does not become normal. Under ideal conditions, great improvement, however, may be maintained. The **worst cases** are rare in this country; but little improvement in them is probable.

The **diagnosis** is based on the temperament, the symptoms, the stigmata. Care must be used not to disregard other illness because of associated hysteria.

Treatment.—A quiet life, free from excitement, with moderate work, careful diet, and other general measures is essential.

Weir Mitchell treatment often helps. It embraces isolation, rest in bed, massage, electricity, non-stimulating entertainment by the nurse only, a very generous but careful diet. Later, active exercise, according to the patient's strength. This treatment allows of no compromise with friends or relatives. The isolation must be maintained for several weeks or months.

The etiologic factors should be avoided, ameliorated or counteracted when possible. Some patients are capable of considerable growth of moral stamina and self-control. For the convulsions and coma, ice or cold water on chest and face, smelling salts or the faradic brush may be tried. Apomorphin, hypodermically, is excellent. The various visceral neuroses need their proper treatment. Drugs: valerian, asafetida, musk, camphor, iron, strychnin, and many others are often used.

QUESTIONS.

Give the etiology of hysteria.

Give the symptoms, mental, sensory, motor, vasomotor, etc., the stigmata.

Describe hystero-epilepsy and differentiate it from epilepsy.

What is the course of the disease and the treatment?

NEURASTHENIA.

Etiology.—**Heredity.**—The parents may show a marked neuropathic taint, or are merely nervous, or weak physically. Neurasthenia is acquired easily, sometimes inevitably, by such persons, or less easily by others who make too great demands upon their nervous energy in worry about business, family or school affairs, or about nothing; in too hard a mental occupation; or in an ordinary occupation when the bodily strength is reduced, as after influenza, typhoid with syphilis, anemia, wasting diseases, enteroptosis, too hard physical work, tea, coffee, alcohol, tobacco, morphin, sexual excess. Cases due to trauma are described on p. 433. On the whole, neurasthenia is much more common in cities, in adult life or adolescence, and perhaps in males.

Symptoms.—These are given by the patient in elaborate detail and are very varied, but all are due to undue fatigue of nerve centers from the excessive use of nervous energy.

Cerebral or Psychic Symptoms.—An increasing inability to do mental work accurately and conclusively; loss of power of attention; exhaustion after moderate or slight mental effort; a sense of fulness and pressure in the head, often headache; often restless sleep or insomnia and a sense of fatigue in the morning. The patient is very often very low-spirited, perhaps hypochondriac, and fears insanity. Women are emotional as in mild hysteria. Vertigo is not rare. There is a dread of meeting people, fear of open places or crowds or of journeys, etc. At such times the patient may tremble, sweat, feel weak or faint and can not move: the heart palpitates and great prostration may follow for several hours. The thoughts may run on actively, and quite beyond control, especially in bed. The patient is apt to talk and think about his many ills and himself. He has a striking inability to come to a definite decision about anything.

Motor Symptoms.—There is the same undue fatigue, often coming on abruptly with a feeling of sudden exhaustion. A fine tremor is common. Local twitching and restlessness may occur. Patients may feel weak enough to stay in bed. There may be a distinct incoördination of movement which may render walking difficult. The muscles are often tense and not relaxed during rest. This shows in a tense expression and in the attitude and movements of the patient. The reflexes vary, but are often increased.

Sensory Symptoms.—Paresthesia, such as numbness and itching, and irregularly distributed hyperesthesia are common. Anesthesia does not occur. Slight discomfort may be called pain, and never escapes attention. There may be tenderness over the vertebræ and back, with aching, especially in the loins or limbs. Various neuralgias are common. *Special senses:* The eyes show the same fatigue. Vision is blurred, the eyes are painful. The pupils may be unequal or dilated, but the reflexes persist. There may be hyperacusis or painful hearing.

Other symptoms are common, such as palpitation, tachycardia, arrythmia, cardiac pain. Even pseudo-angina may occur. Vasomotor symptoms are flushing, local heat, especially in the head; sweating, local or general; often a capillary pulse; throbbing vessels, especially in the head or epigastric region (aorta), which may be very distressing to the patient. On the other hand, they very often have pale, cold and clammy hands and feet. Gastro-intestinal neuroses are important and common. Genito-urinary symptoms: In men there may be irritable weakness of the sexual organs, temporary impotence, exhaustion after coitus: spermatorrhea and testicular neuralgia are common. In women there are often ovarian or other genital neuralgias, sacral pain, backache, painful or irregular menstruation. The urine often contains an excess of urates, oxalates or phosphates. Frequent micturition is common. The general appearance and nutrition of the patient varies, usually he is thin and pale, looks run down, and has a tense expression. Occasionally they look ruddy and well.

Various types of neurasthenia are described: the cerebral, spinal, gastro-intestinal, sexual, lithemic, and others. In each the most prominent symptoms are suggested by the name.

Course.—The onset is slow and gradual and the disease shows all grades of severity. It may last any length of time and does not shorten life. Marked improvement is usually possible, or even a complete cure, but relapses are apt to occur with a repetition of the causes.

Treatment.—**Preventive.**—This is often more difficult because of the example of the neurasthenic parent, and in such cases a child is usually better off in a boarding school in the country. The child should be taught self-control by precept and, especially, by example; taught to carry a task to a definite end, and to then accept the result as final, and not to repeat the work time and time again; to disregard his trivial ills; to cultivate regular habits of exercise, eating, studying, and sleeping. At puberty, especially in girls, there is apt to be an increase of nervousness. In this case the child should be taken out of school and sent to the country for a year or two. Throughout life persons of neurotic tendency should observe unusual care in exercising moderation in eating, mental exertion, in tea, coffee, alcohol, tobacco; to take cold baths and regular exercise; to exercise during vacations, but always within the limits of their strength.

In an Attack.—A cure will follow if the patient can be taught not to waste his energy in apprehensions and worry, and to forget himself. His time should be occupied with easy, unstimulating tasks, including exercise in proportion to his strength. A protracted absence from business is desirable, the time being spent in the country or in travel. When he is tired he should rest and relax. Mild cases of short duration often do very well with hydrotherapy and a reasonable life. Severe cases, especially in women, often do very well with the Weir Mitchell treatment. Drugs are bad, because patients are apt to expect them to effect a cure. Nevertheless, glycerophosphates of sodium, calcium and iron, strychnin, and in anemic persons, iron often help.

Special symptoms, such as constipation, anorexia, nervous indigestion, etc., need treatment. Sleeplessness is best treated by hydrotherapy—occasionally it may need drugs, such trional, but morphin, chloral, and alcohol are hardly justifiable.

QUESTION.

Give the etiology, symptoms, prognosis, and treatment of neurasthenia.

THE TRAUMATIC NEUROSES.

(**Traumatic Hysteria, Traumatic Neurasthenia, Railway Spine, Railway Brain.**)

Etiology.—These conditions follow an accident or mental shock in a neurotic person, with or without physical injury. A slight fall or a great nervous shock with exposure may cause them.

Symptoms.—(1) **Simple Traumatic Neurasthenia.**—This may develop several weeks after the exciting cause. The symptoms are those of a rather typical neurasthenia, with marked depression, sometimes even melancholia. Pain in the back or head and rigidity of the back are rather common symptoms.

(2) **Traumatic Hysteria.**—Beside the neurasthenic symptoms there are the symptoms of actual hysteria, with the stigmata, sensory disturbances, often unilateral, a marked tremor, paralyses and contractures of one or more limbs.

(3) Cases with **symptoms of organic disease of the brain and cord:** Some cases have actual lesions, such as pachymeningitis, local arteriosclerosis, punctate hemorrhages, degeneration, especially of the lateral column, or multiple sclerosis of the brain and cord. The symptoms pointing to an actual lesion are optic atrophy, involvement of the sphincters, especially of the bladder; exaggerated reflexes, paresis, and tremor. These patients may die of the lesion.

The usual cases are pure neurosis and are not rare. They show various motor and sensory symptoms, as in hysteria.

The **diagnosis** is made by finding a cause, recent or somewhat remote, for the neurasthenic and hysteric symptoms. The neuroses of the last group can not always be diagnosed from the cases of organic lesions until optic atrophy develops.

Prognosis.—These neuroses almost always disappear, but perhaps not for years. The patients usually recover promptly after a favorable verdict in a suit for damages.

QUESTION.

Give the symptoms of the traumatic neuroses.

PERIODIC PARALYSIS.

Etiology.—This is usually a family disease, but isolated cases have occurred.

Lesions are present, at least in some cases. There is a fatty degeneration and vacuolization of some muscle fibers. It is supposed to be an auto-intoxication, attacking the muscle fibers.

Symptoms.—An apparently healthy person is attacked, often in sleep. There are loss of strength in the muscles, flabbiness, no reaction whatever to galvanic or faradic electricity, the deep reflexes are absent, the cutaneous reflexes present or absent. The symptoms begin in the feet, extend up the leg and thigh, later involve the arms and the trunk. The diaphragm and cranial nerves escape. There are no trophic, vasomotor or sensory symptoms. After a few hours or a few days the patient gets well. The attacks recur, sometimes with perfect regularity, at intervals varying from one day to a year. They usually cease after a few years and do not kill.

Treatment.—Quinin and potassium citrate may cure.

QUESTION.

Give the causes and symptoms of periodic paralysis.

ASTASIA-ABASIA.

A pure neurosis occurring in hysteria, neurasthenia, etc. Astasia is an inability to stand, abasia an inability to walk. All other movements of the legs, the electric reactions, and the muscular strength are wholly unaffected. The incoördination is evident only in standing or walking or both. The patients usually get well.

Treatment.—Electricity and causal treatment.

QUESTION.

Give the etiology and symptoms of astasia and abasia.

VASOMOTOR AND TROPHIC DISORDERS.

RAYNAUD'S DISEASE (Symmetrical Gangrene).

Etiology.—It is more common in females in the first half of adult life, and sometimes is hereditary. The most important factor is exposure to cold. It may follow intense emotion. It is not infrequently associated with tuberculosis, disease of the heart and arteries, scleroderma, and various neuroses.

Symptoms.—There are three stages: (1) **Local syncope** (dead fingers). The fingers, and often toes, are white, cold and numb with impaired sensation. There is a spasm of arterioles, and probably of venules also. (2) **Local cyanosis** or local asphyxia. The venules relax first, the fingers swell, become cyanotic, sometimes dark purple, and may be very painful. This may occur without the first stage. (3) Local or **symmetrical gangrene** or necrosis may follow if the cyanosis lasts several hours or days. There is intense pain, with necrosis usually of the skin only, sometimes of deeper tissues also. The fingers, toes, and tips of the ears on both sides, and the tip of the nose, are the common sites. The attacks are paroxysmal, usually in winter, and recur at varying intervals. The disease does not kill, but may cause great suffering. After years it may cease. Hemoglobinuria may occur during or instead of an attack following exposure. Mental torpor, transient unconsciousness, epileptic attacks, transient hemiplegia, and other symptoms may accompany an attack.

Treatment.—In the attack, galvanism is used, the extremity and the cathode being immersed in salt water, with the anode on the spine. Massage is used. Nitroglycerin is given in the first stage. The limb is elevated in the later stages. Morphin may be needed for the pain.

QUESTION.

Give the etiology and the symptoms of Raynaud's disease.

Erythromelalgia (*red neuralgia*) is a rare affection of the feet or hands. It is usually found in one or both feet. There are redness, pain, often intense, some swelling and local heat. All symptoms are more marked if the limb hangs down. It is apt to be chronic, to be aggravated by heat and relieved by cold. The treatment consists of cold and elevation of the limb.

QUESTION.

Give the symptoms of red neuralgia.

Angioneurotic edema (*acute circumscribed edema*) is often a family disease. It is marked by sudden, soft, edematous swellings in the eyelids, on the face, hands, genitals or elsewhere. These may be preceded by itching, heat or redness. They may rarely be accompanied by gastro-intestinal crises, cardiac pain or hemoglobinuria. The edema may attack the throat and larynx and be fatal, otherwise it is not dangerous and passes away in a few hours.

QUESTION.

Give the symptoms of angioneurotic edema.

Facial hemiatrophy is a rare condition of unknown etiology, that is not congenital. There is atrophy of the structures of one-half of the face; first of the soft tissues of the face, the tongue, and the palate; later, of the jaw and malar bones, and sometimes the nose and ear cartilages. There may be sympathetic nerve disturbances (pallor, dilated pupil, anidrosis), also defective sensation, perhaps fewer teeth. The eye looks sunken from atrophy of orbital fat.

ACROMEGALY.

Etiology.—This is a rather rare disease that begins in the first half of adult life.

Lesions.—The pituitary body is enlarged from hyperplasia or neoplasm. The thyroid may be large or atrophied. The thymus often persists. Other lesions are mentioned below.

Symptoms.—There is a slow, progressive enlargement of the face, hands, and feet, involving bones, cartilages, and soft tissues. In the hands and feet this is shown by distinct enlargement in all directions, especially in breadth and thickness, giving the spade-like hands. Otherwise they are not deformed and function persists. The eyelids and tongue enlarge, the features become coarse, the face projects beyond the cranium and looks very large; the lower jaw is very prominent, the teeth are separated. The larynx may be enlarged, the voice deep and rough, speech may be thick. Cerebral symptoms are usually present, chiefly a headache, which may be very severe and constant. Temporal hemianopia (from pressure on the chiasm) or optic atrophy may occur. There is loss of sexual function and in women amenorrhea. Kyphosis and enlargement of the bones of the trunk may appear. The disease lasts many years and treatment is unsuccessful.

QUESTION.

Give the lesion and symptoms of acromegaly.

Osteitis deformans is a chronic, progressive enlargement and softening of the long bones and the cranium, but not of the facial bones. The limbs are curved and deformed, pain is marked at first or throughout, the mind is not affected.

Hypertrophic pulmonary osteo-arthropathy is a rare condition, associated with chronic lung disease, especially chronic bronchitis, emphysema, empyema, tuberculosis, or bronchiectasis. It involves only the hands and feet and lower part of the forearms and legs. The bones are thick and curved, the nails are thick, large, and curved like talons.

Scleroderma is rather more common in females and in adult life. (1) **Circumscribed form:** Patches, one inch or more in diameter, are observed. At first they may be hyperemic; but later the skin is white, thick, leathery. It may become pigmented or a leukodermatous patch. Sensory changes are unimportant. The most common sites are on the breast or neck. The spots may develop rapidly or slowly, and last for weeks or years. (2) **Diffuse form:** This involves extensive areas on the head, trunk or limbs, sometimes it is nearly universal. The skin is stiff, thick, adherent to the deeper

structures, smooth, dry, and glossy. It interferes serio
with movement. There may be cyanosis and tachycar
Sensory symptoms are unimportant. It develops slowly,
last many years, but sometimes disappears. *Treatment*
Tonics, warm climate and clothing, warm baths and frictio

QUESTION.

Give the symptoms of scleroderma.

DISEASES OF MUSCLES.

MYOSITIS.

Polymyositis (*Dermatomyositis*) is a rare and probably infectious condition. **Lesions.**—The muscle involved is pale, swollen, and watery. Microscopically, there is granular, fatty, and hyaline degeneration of the fibers, with vacuolization. There is some hyperplasia of the connective tissue. **Symptoms.**—There may be a chill, fever, perhaps a large spleen. The affected muscles are very painful, hard, and tender. The skin over them may be edematous, or show rashes. The disease may kill, in from two to four weeks, by exhaustion; or may end in slow recovery after one to three months or more. The diagnosis from trichinosis is made by the examination of an excised bit of muscle. (The eosinophile count in this disease is not known.)

Progressive Ossifying Myositis.—This, also, is rare. It involves the muscles of the back and back of the neck; later, the other neck muscles and those of the chest; finally, those of the limbs and of the lower jaw. At first, there may be fever. The involved muscle is painful; the skin over it may be red and edematous, the muscle grows firmer and harder—first from an infiltration with dense fibrous tissue; later this becomes cartilaginous and finally ossified in plates or irregular masses. The muscles finally become perfectly rigid and hard. They are attacked one after the other. The disease lasts ten or twenty years.

QUESTION.

Give the symptoms of polymyositis and ossifying myositis.

MYOTONIA (Thomsen's Disease).

Etiology.—This is usually hereditary and begins in childhood, even infancy.

Symptoms.—It is a muscular disturbance, shown only with voluntary movements. The muscles contract slowly, but well; voluntary relaxation appears only after a few seconds' delay and is very gradual. In walking, after a few slow, difficult steps, the muscles limber up and the patient walks well enough. It involves the limbs usually, rarely the face and larynx. The muscles show the *myotonic electric reaction*—the current induces a slow, protracted contraction, with wave-like movements, passing from cathode to anode. The muscles are unusually large, but not unusually strong. The disease is incurable, does not shorten life, and may grow less troublesome in later years.

QUESTION.

Give the symptoms of myotonia.

THE MUSCULAR DYSTROPHIES.

General Points.—Etiology.—A majority of cases show direct heredity or family involvement. It is, perhaps, more common in males, but is more often transmitted through the mother. It develops before puberty, as a rule, and but very rarely after twenty-five.

Lesions.—In some cases the nerves and anterior horn cells have been found to be degenerated, but this is rare. The chief change is in the muscles themselves. The *muscle fibers* at first are larger and round, perhaps fissured. The nuclei are somewhat increased. Later, sometimes almost from the start, the fibers atrophy, vacuoles and fissures appear, and the nuclei are much increased. Along with this there is *connective-tissue hyperplasia.* Sometimes the new connective tissue contains fat and may for a time even increase the size of the muscle (pseudohypertrophy); in other muscles it just about compensates for the atrophy; while still others grow smaller from the start. In time all of the muscles are small and fibrous. Only certain muscles are apt to show pseudohypertrophy, and all stages of atrophy may be found in the same muscle. The connective tissue may contract and shorten the muscle with consequent deformity. Later, the strong, opposing muscles may counteract this and even cause contracture with opposite deformity.

Symptoms.—The changes usually begin in the calves, trunk, face, or shoulder girdle, not in the hands or feet. There are no fibrillary twitchings, no reaction of degeneration, but the electric excitability and the deep reflexes progressively decrease as the muscle fibers disappear. Sensory symptoms are absent. The sphincters are not involved. The disease is progressive. The patients become bedridden in the course of years, and they die of exhaustion or more commonly of an intercurrent disease, especially pneumonia or bronchitis.

The **diagnosis** must be made from other conditions with muscular paralysis or wasting—for example, cerebral lesions, progressive spinal muscular atrophy, progressive neural muscular atrophy, and multiple neuritis.

There are three chief **clinical types,** but intermediate and atypical cases occur.

(1) **Pseudohypertrophic Muscular Paralysis.**—This is many times more common in males. It is noticed when the child learns to walk, or at least before six years. There are at first weakness and awkwardness of the legs, with stumbling and difficulty in climbing stairs and in rising. Some muscles tend to show pseudohypertrophy, and look unusually large. This is common and very marked in the calves and the infraspinati; occurs to a varying degree in the extensors of the knee, the glutei, the lumbar muscles, the supraspinati, and the deltoid. Others atrophy markedly—the latissimus dorsi, the lower part of the pectoralis major. Others are spared, especially the muscles in the hand and, usually, the trapezius, the muscles of the face, neck, and the forearm.

In rising from the floor the child gets on his hands and knees; then on his hands and feet; then he works his hands up his legs (climbs up his legs), and so rises, straightening himself at last with a jerk. He can not stand on tiptoe. When sitting there is marked kyphosis of the whole spine; when standing, a marked lumbar lordosis and a compensatory kyphosis above.

The contracting connective tissue of the calf muscles may lead to talipes equinus. Later the pull exerted by the sound muscles may counteract this.

(2) **Juvenile Type of Idiopathic Muscular Atrophy** (Erb).—(*Scapulohumeral Type*).—This appears at puberty in either sex, and usually in more than one member of a family.

Symptoms.—It begins with atrophy of the muscles of the shoulder girdle, upper arm, chest and back. The lower extremities are involved much later. It spares the muscles in the hand and the deltoid. The trapezius is often attacked, and then always in all its parts.

(3) **Infantile Type** (Duchenne) or *Facio-scapulo-humeral Type of Idiopathic Muscular Atrophy* (Landouzy-Déjérine).—This begins at the third or fourth year, or at puberty. First, there is atrophy of the eyelids and lip muscles, so that neither the eye nor the mouth can be closed; then of the rest of the face, giving the facies myopathica—a perfectly blank expression. The ocular muscles and the tongue escape. Later, perhaps only after years, it attacks the shoulder girdle and the arm muscles, and finally may spread to the back and legs.

The **treatment** consists merely of general management, massage, moderate active exercise when possible, orthopedic appliances.

QUESTIONS.

Give the etiology and symptoms of the muscular dystrophies in general, also of the three chief types.

Give the differential diagnosis of these conditions from neural muscular atrophy, spinal muscular atrophy, multiple neuritis, cerebral paralyses.

Give the prognosis and treatment.

ARTHRITIC MUSCULAR ATROPHY.

Sometimes following acute or chronic arthritis there is a rapid atrophy of the related muscles, too marked for mere disuse. This affects the extensors chiefly. The atrophy sometimes persists for a considerable time after the joints recover. The electric excitability is lessened, the mechanical excitability and deep reflexes may be increased, and rarely fibrillary twitchings are seen.

Treatment.—The arthritis should be carefully treated. Besides this, gentle massage, electricity, and moderate exercise when feasible may help the muscles.

QUESTION.

Define arthritic muscular atrophy.

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